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ENCEPHALITIS COMPLICATING MEASLES

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During the five and one-third years between January 1936 and May 1, 1941 there were admitted to the Queensboro Contagious Pavilion 29 patients with encephalitis which followed acute contagious diseases of childhood. In the greatest number of these, 20 (69 per cent), the infection followed measles; in 4 (14 per cent), pertussis; in 4 (14 per cent), mumps, and in 1 (3 per cent), chickenpox. Of the 20 patients with encephalitis complicating measles, 16 (80 per cent) were admitted to the hospital within the period of less than three and one-half months from Jan. 29, 1941 through May 1, 1941. This period corresponded to one of increased incidence of measles.

ETIOLOGY

Inasmuch as an appreciable number of instances of measles are not reported, one can only speculate on the incidence of encephalitis as a complication of this disease. Neal [1] has stated that before epidemic encephalitis made its appearance in this country one rarely observed cases of encephalitis following measles. Ford,[2] quoting Boenheim, stated that among 5,940 patients with measles treated in various hospitals in Berlin between 1905 and 1925 0.4 per cent had complications involving the nervous system. Of these, 8 had serous meningitis; 6, encephalitis, and 11, unexplained convulsions. Geiger and Sappington [3] reported 13 cases of encephalitis associated with measles in two epidemics of measles with a total of 17,528 cases—an incidence of about 0.08 per cent. Peterman and Fox [4] reported 13 cases of encephalitis complicating measles out of a total of 15,001 cases.

In our group of 20 patients there were 12 boys and 8 girls, the ages ranging between 2 and 9 years and the average age being 5.7 years.

There are three views concerning the causation of encephalitis with measles: (1) that it is due to the virus of measles; (2) that it is an allergic or anaphylactic phenomenon, and (3) that it is caused by an unknown virus separate and distinct from that of measles.

PATHOLOGY

The pathologic process in the brain and the spinal cord has been described by Moore and McCordock,[5] Malamud,[6] Ziskind and Schattenberg,[7] Ferraro and

From the Contagious Pavilion, Department of Pediatrics, Queens General Hospital, Jamaica, N. Y., Dr. Henry A. Reisman, Director.

1. Neal, J. B.: J. Pediat. **8**:521, 1936.
2. Ford, F. R.: Bull. Johns Hopkins Hosp. **43**:140, 1928.
3. Geiger, J. C., and Sappington, E. E.: Arch. Pediat. **57**:313, 1940.

(Footnotes continued on next page)

Scheffer [8] and others. The most frequent lesions observed are congestion and small hemorrhages about the blood vessels. Moore and McCordock [5] found perivascular demyelination the most outstanding pathologic change in the majority of recorded cases. The lesions most frequently involve the white matter, but in some cases there is involvement of the gray matter. The perivascular cuffing is chiefly by microglial elements. Rivers [9] has stated that no known virus acting directly on the central nervous system produces a perivascular demyelinating type of encephalitis.

Fig. 1.—Section of brain showing engorgement of vessels with perivascular cuffing. Hematoxylin and eosin stain; low magnification.

4. Peterman, M. G., and Fox, M. J.: Postmeasles Encephalitis, Am. J. Dis. Child. **57**: 1253 (June) 1939.

5. Moore, E., and McCordock, H. A.: Encephalomyelitis and "Hemorrhagic Virus Pneumonia" Complicating Measles, Arch. Neurol. & Psychiat. **32**:560 (Sept.) 1934.

6. Malamud, N.: Lissauer's Dementia Paralytica: Study of Its Pathogenesis, Arch. Neurol. & Psychiat. **38**:27 (July) 1937.

7. Ziskind, J., and Schattenberg, H. J.: Postmeasles Encephalitis, Arch. Path. **27**:128 (Jan.) 1939.

8. Ferraro, A., and Scheffer, I. H.: Toxic Encephalopthy in Measles, Arch. Neurol. & Psychiat. **27**:1209 (May) 1932.

9. Rivers, T. M.: J. Pediat. **8**:518, 1936.

Necropsies were done on the 2 patients that died in our group of 20. Abstracts of the pertinent pathologic data follow:

CASE OF R. B.—The brain was bulging through an opening in the dura, which was taut over the intracranial contents. The cortical tissue was edematous, and the sulci were definitely narrowed and linear. The gyri were flattened. The cortical tissue was congested; the cerebral nerves were intact. No obvious hemorrhages were noted. When sections were taken the cut surface showed congestion, prominent vascular markings appearing as bleeding points. The cortical tissue stood out prominently in contrast with the diffuse congestion.

Sections of the brain tissue stained with hematoxylin and eosin showed marked engorgement of both large and small blood vessels, which were surrounded by a mantle of cells, for the

Fig. 2.—Section of brain showing engorgement of vessels with perivascular cuffing. Hematoxylin and eosin stain; medium high power magnification.

most part in layers two or three cells thick, in which round cells and large mononuclear elements were prominent. No marked demyelination was evident in the routine preparations. An increase of glial nuclei was noted about some of the involved vessels. Other vessels showed no changes. Intravascular thrombi were observed.

The final anatomicopathologic diagnoses were as follows: encephalitis associated with measles, cutaneous rash, lymphoid hyperplasia, hyperplastic splenitis, acute bronchitis, bronchopneumonia and bilobed spleen.

CASE OF L. S.—When the dura was incised the brain substance was found to expand beyond the cut edges of the membrane. The brain was found to be flaccid, and the cortical convolutions to be markedly flattened, with the sulci obliterated, appearing only as linear

depressions. The surface of the brain showed a fine, diffuse but intense congestion. Sections of the brain displayed intense congestion, particularly of the cortex, the larger vessels being prominent and having considerable blood content. No areas of macroscopic hemorrhages were noted.

The blood vessels of the brain were intensely engorged and were surrounded to a certain extent with a mantle of rather pale-staining mononuclear cells. Some perivascular demyelination could be observed in sections stained with hematoxylin and eosin. Other vessels revealed intense, closely placed perivascular cells as a complete encircling cuff. The glial elements did not seem to be markedly increased about most of the involved vessels. Some, however, showed increase of glial nuclei in adjoining and nearby areas.

The final anatomicopathologic diagnoses were as follows: encephalitis associated with measles, acute bronchitis, bilateral patchy atelectasis, pulmonary congestion, generalized lymphoid hyperplasia with measles cells, petechial hemorrhages into follicles of the ileum and ileal mucosa.

SYMPTOMATOLOGY

The manifestations of the disease show considerable variation. The symptoms may vary from headache and fever in 1 patient to deep coma with a multitude of abnormal neurologic findings in another.

Onset.—The outstanding feature of the onset of the disease in most of our cases was its suddenness, with the alarming symptoms referable to the central nervous system. The interval between the onset of the measles rash and that of the encephalitis was from two to six days except in 1 case, in which the interval was eleven days. In this case pneumonia had preceded this condition, and there was some question as to whether we were dealing with an embolus complicating the pneumonia, inasmuch as the patient presented hemiplegia (left side). The child recovered and showed only a slight weakness of the left hand one month after the illness. In all 20 cases the average interval between onset of the rash and that of the encephalitis was four and three-tenths days.

Temperature.—All our patients had some fever, the temperature varying from a slight rise to 108 F. The duration of the fever was from one day to several weeks. The two highest temperatures noted were 107 and 108 F. Both patients with such abnormal temperatures died, one within three hours of admission to the hospital and the other twenty-four hours after admission.

Stupor and Coma.—Fifteen of the 20 patients became either stuporous or comatose. Their condition varied from a stupor from which the patient could be aroused to a state of profound unconsciousness. There was considerable variation in the duration of the stupor and coma, this state lasting from several hours to several weeks.

Convulsions.—Twelve of our patients had convulsions or twitchings. In some these were limited to an extremity of the body, and in others they were generalized. All responded to adequate sedation.

Other Symptoms and Signs.—These included stiffness of the neck and back, hyperesthesia, headache, vomiting, absence of superficial and deep reflexes, disorientation, confused mental states, irritability, hallucinations, aphasia, papilledema, incontinence, defects of speech and paralyses.

LABORATORY DATA

Blood.—Results of examination of the blood were of no special significance. The total white cell count varied between 5,800 and 18,400, and the polymorphonuclear neutrophil count between 53 per cent and 82 per cent. Peterman and Fox [4] in their group of cases emphasized the fact that polymorphonuclear leukocytosis was a consistent feature of this condition, reporting white blood cell counts between 12,600 and 26,000.

Data in Twenty Cases of Encephalitis Complicating Measles

				1. Summary of Clinical Findings				2. Spinal Fluid Findings				
No. of Case	Patient	Age, Years	Sex	Date of Admission	Date of Discharge	No. of Days After Rash	Salient Features	Pressure, Mm. of Water	Cells	Sugar, Mg. per 100 Cc.	Protein, Mg. per 100 Cc.	Chlorides, Mg. per 100 Cc.
1	G. Q.	4	M	5/18/36	5/22/36	?	Temp. 102 F.; fading measles rash; moderate rigidity of neck; absence of deep reflexes; coma	.....	45 lymphocytes	.....	..	...
2	B. D.	6	M	5/ 6/38	5/11/38	3	Temp. 102.6 F.; headache; neck rigid; abdominal reflexes and knee jerks absent	Increased	150, 100% lymphocytes	.....	0	...
3	E. B.	6	F	5/22/38	5/29/38	6	Temp. 105 F.; convulsions; coma; neck rigid; abdominal reflexes absent	9 mm. Hg	150, 100% lymphocytes	168.6	0	753
4	H. K.	6½	F	4/13/38	4/28/38	4	Temp. 104.4 F.; convulsions; drowsiness; neck rigid; Kernig sign present; left knee jerks absent	Slight increase	16	111.1 88.2	0	706 754
5	L. S.	5	F	1/29/41	Died 1/29/41	3	Temp. 108 F.; convulsions; coma; reflexes absent; death three hours after admission	.....	40, 100% lymphocytes	156	0	...
6	E. G.	6	F	2/ 5/41	3/31/41	4	Temp. 104 F.; convulsions; coma; fading rash; reflexes absent; neck rigid; aphasia	.....	105, 89% lymphocytes	81 71 90	37.5 47.5	663 742
7	S. O.	4	M	1/22/41	1/28/41	2	Temp. 103 F.; convulsions; coma; fading rash; fever one day; rales in chest	.....	47, 45% lymphocytes	Increased	Increased	...
8	A. K.	5	F	2/28/41	3/10/41	4	Temp. 101.5 F.; convulsions; coma; reflexes absent	Increased	93, 100% lymphocytes	144	33	...
9	B. S.	7	F	3/ 2/41	3/ 6/41	5	Temp. 101.5 F.; convulsions; coma; cyanosis	Low	23, 83% lymphocytes	93	0	...
10	J. B.	3	M	3/ 6/41	4/ 5/41	11	Temp. 104 F.; convulsions; coma; left hemiplegia; reflexes absent; preceded by lobar pneumonia	.....	10	80 68	33	706 728
11	J. C.	8½	M	3/21/41	4/ 5/41	6	Temp. 102.6 F.; neck rigid; Kernig and Brudzinski signs present; irritable	240	296	71	..	...
12	R. L.	7	M	3/26/41	.....	3	Temp. 101.6 F.; stuporous; headache; leg pains; Kernig sign present; abdominal reflexes absent	120	Traumatic	49	..	...
13	R. B.	2½	M	3/28/41	Died 3/29/41	3	Temp. 107 F.; convulsions; coma; died 24 hours after admission	Low	4 lymphocytes	122	..	804
14	S. G.	6	M	3/30/41	5/28/41	2	Temp. 98 F.; coma; delirium; rigid neck; superficial reflexes absent	180	11 cells, 90% lymphocytes	69 77	30 30	752
15	S. Z.	7½	M	4/ 4/41	5/12/41	5	Temp. 104 F.; headache; vomiting; neck rigid; hyperesthesia; abdominal reflexes absent	.....	360 lymphocytes	67 62 101	26.5	752
16	M. M.	5½	M	4/ 8/41	4/13/41	3	Temp. 102 F.; drowsiness; headache; neck rigid; Kernig sign present	120	1,260 lymphocytes	101	72.5	...
17	L. W.	5	F	4/21/41	4/25/41	6	Three convulsions; coma; results of neurologic examination negative	120	4 lymphocytes	.....	..	...
18	R. S.	8	M	4/25/41	5/11/41	5	Temp. 104 F.; abdominal pain; stiffness of back; results of neurologic examination negative	.....	945 cells, all polymorphonuclears	65	..	673
19	C. P.	9	F	4/29/41	5/14/41	3	Temp. 104 F.; hemorrhagic rash; coma; convulsions; neck rigid; abdominal reflexes absent	225	180 cells, 70% lymphocytes	64 66	55 53	678 690
20	D. H.	2	M	5/ 1/41	5/ 6/41	3	Temp. 101 F.; convulsions; coma; cyanosis; fading rash	.....	60 cells, 90% lymphocytes	..	..	...

Spinal Fluid.—Encephalitis, being primarily an involvement of nerve tissue, does not necessarily cause changes in the spinal fluid. However, the majority of patients do show signs of meningeal involvement. In 16 of our 20 patients the spinal fluid showed an elevated cell count, varying between 11 and 1,260 cells. As high a count as this is unusual. In this patient the fluid presented a ground glass appearance. Within a few days it became clearer, and the cell count dropped to 90. In most of our patients the fluid showed a preponderance of lymphocytes. However, in 1 there was a pleocytosis of 945 cells, all being polymorphonuclear leukocytes.

It is generally stated that the sugar content in the spinal fluid in encephalitis complicating measles is either normal or increased. Our findings concur with this statement. Twenty-three quantitative determinations of sugar were made on 15 of the 20 patients. Fourteen of these 15 patients had an elevated sugar content of the spinal fluid, ranging between 65 and 168.6 mg. per hundred cubic centimeters.

The protein content of the spinal fluid is as a rule normal. In only 2 of our patients was it increased. The highest value we obtained was 72.5 mg. per hundred cubic centimeters. This was observed in the patient whose spinal fluid had a ground glass appearance, and contained 1,260 cells, all of which were lymphocytes.

The pressure of the spinal fluid was not constant, being normal, increased or decreased.

The amount of chlorides in the spinal fluid was within normal limits.

DIAGNOSIS

The majority of patients have either a fading eruption of measles or a characteristic discoloration following the eruption. The history of measles, the sudden invasion of symptoms pointing to involvement of the central nervous system, usually two to six days after the onset of the measles rash, the varied symptoms and the changes in the spinal fluid should point to a diagnosis of encephalitis complicating measles. Other conditions which may have to be considered are meningitis, poliomyelitis, epidemic and other types of encephalitis, cerebral embolus, tumor and abscess of the brain and meningococcemia.

SEQUELAE

Of the 18 children who survived, 2 were examined four years after the illness, and 8 one year after the attack of encephalitis. Eight of the 10 patients examined were entirely normal. The other 2 patients showed definite changes in personality without mental deterioration. The incidence of sequelae does not seem to have any relation to the severity of the attack of encephalitis.

REPORTS FROM THE MENTAL HYGIENE CLINIC ON THE TWO CHILDREN

CASE OF E. G.—A girl aged 6 years was reported by her mother to have been "a little nervous" before she had measles. She ascribed this to the fact that the father paid more attention to the other child than to the patient.

Since having measles, the child had experienced an episode of confusion at home. She said there was a man in the corner and asked her mother to chase him away. Her mother found the child overactive and overtalkative.

Her mother also noticed that the child confused colors. The results of the Ishihara test were not conclusive on account of the difficulty of the patient in giving attention.

As a result of psychometric tests given on April 17, 1941 the child was rated as one of average intelligence but with mental responses suggestive of a disturbance of organic type.

The child has since been treated regularly in the mental hygiene clinic with play therapy. Under this treatment she showed continuous improvement in her general behavior.

On June 27, 1941 another psychometric examination was made, with the following results: 1. In Koh's block design test she was unable to grasp the concept of total configuration. 2. In the Rorschach test she showed the following evidences of organic disturbance: perplexity, insecuritiy and uncertainty; number of responses less than 15; large number of poor forms, and absence of movement and motor tendencies.

CASE OF S. G.—A boy aged 6 years was considered a "nervous child" before he had measles. He was described by his parents as "very active, bad, and disobedient." He was regarded as a bright child.

His parents stated that after he had measles he became very stubborn, stamped his feet, "kicks, bites, fights and uses very bad language" and hit his mother.

When seen in the clinic, the patient showed extreme overactivity of the type that one usually finds in postencephalitic behavior disorders in children. He cried out for no apparent reason and was extremely irritable.

This child also was treated with playroom therapy, but since this was given a long distance from his home, he could come only a few times.

According to psychometric tests, this child was functioning at a moron's level with an intelligence quotient of 63. However, results of the Rorschach tests showed that his original endowment was not only good but actually superior. The tests indicated clearly an organic involvement.

There has been a remarkable improvement in this child, and when he was last seen in the clinic the mother reported that he was going to school and that he had a grade of "A" in conduct but "D" in work. However, according to the mother, he is still "very nervous" and still has temper tantrums.

This child is to return to the mental hygiene clinic for further treatment.

Ford [2] abstracted histories of 125 cases and noted mental defects in 22. He stated: "About sixty-five per cent of those who survive show residual symptoms: weakness in thirty per cent, ataxia in twelve per cent, mental defect or personality changes in seventeen per cent and epilepsy in five per cent." Ford [2] also stated that the commonest mental residuum of encephalitis complicating measles is a reduction in intelligence. Hamilton and Hanna [10] stated that recovery with residual symptoms can be expected in 40 per cent of those who survive.

MORTALITY

In our group of 20 patients 2 died—a mortality of 10 per cent. Both of these patients were admitted in coma and with hyperpyrexia and died within twenty-four hours after admission to the hospital with what appeared to be an overwhelming invasion of the central nervous system. The mortality rate for encephalitis associated with measles has been variously reported between 10 per cent and 35 per cent, most reports showing a mortality of 10 to 20 per cent.

TREATMENT

Treatment was entirely symptomatic. Sedation was used for restlessness, twitchings and convulsions. In a few cases spinal fluid was withdrawn to relieve intracranial pressure. In the majority, this was not considered necessary, the spinal tap being done chiefly as an aid in diagnosis and progress. In the cases of more severe symptoms hypertonic medication was given, such as administration of hypertonic solution of dextrose intravenously. Magnesium sulfate was given intramuscularly. To those patients unable to take nourishment by mouth, liquid foods were given by gavage by means of a Levin tube. Vitamins were supplied because of the limited intake of food.

10. Hamilton, P. M., and Hanna, R. J.: Encephalitis Complicating Measles: A Report on Two Hundred and Forty-One Cases Collected from Literature and Forty-Four Additional Cases, Am. J. Dis. Child. **61**:483 (March) 1941.

Several of our patients were given sulfapyridine with little apparent effect. In patients with persistent stupor and high temperature, chemotherapy had no effect on the course of the disease. At times it was felt that chemotherapy might be responsible for the fever. But neither discontinuance nor continuance of chemotherapy seemed to have any effect.

Hamilton and Hanna [10] reported on different types of treatment given to three groups of patients. The first included 18 patients treated symptomatically, with 5 deaths. The second comprised 10 patients treated by one or more of the following methods: administration of convalescent measles serum, administration of human convalescent poliomyelitis serum and immunotransfusion. This group had 5 deaths. The third group, including 16 patients, were given shock therapy by intravenous injection of commercial mixed typhoid vaccine. In this group there was 1 death. As a result of their observations these physicians concluded that shock therapy offers more hope than any other type of treatment available today.

We used shock therapy with typhoid vaccine for a patient who had been in a coma for three weeks. The typhoid vaccine was given every other day, and it was not until after three doses had been given with good fever response that the child began to show signs of clearing mentally.

For another we used measles convalescent serum with apparently no effect.

PROGNOSIS

The prognosis must at all times be guarded and must consider the possibility of sequelae. Hamilton and Hanna,[10] reporting on 241 cases collected from the literature and 44 additional cases of their own, stated that complete recovery can be expected in 40 per cent, recovery with residual symptoms in 40 per cent and death in 20 per cent of the cases.

SUMMARY

Encephalitis complicating measles usually occurs two to six days following the onset of the measles rash, as a rule accompanied by marked and varied symptoms referable to the central nervous system. In the 20 cases of such encephalitis reported here the mortality was 10 per cent. Ten of the patients were examined one to four years after the attack of encephalitis. Two patients were found to have sequelae.

Dr. Alfred Angrist, pathologist, and Dr. Frederic Wertham, chief of the mental hygiene clinic, Queens General Hospital, gave cooperation and aid in the preparation of this article.

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ULTRAVIOLET IRRADIATION OF BLOOD IN THE TREATMENT OF EXPERIMENTAL POLIOMYELITIS

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Ultraviolet rays kill poliomyelitis virus.[1] They also detoxify and inactivate bacteria and toxins. Perhaps blood, which is capable of absorbing ultraviolet rays, would give off secondary emanations which would destroy virus in vivo.

Macaca mulatta monkeys were given poliomyelitis virus intracerebrally and treated as follows: For each treatment, 2 cc. of blood per pound (4.5 Kg.) of body weight was withdrawn from the femoral veins and citrated with one part of 2.5 per cent solution of sodium citrate to five parts of whole blood. It was then run through the irradiation chamber, a labyrinth of beveled plates (to insure thorough turbulation of the blood) and a quartz glass window. Through the latter, held at approximately contact distance from the burner, the ultraviolet rays from a water-cooled ultraviolet generator act on the blood. The blood was then returned to the vein of the monkey through sterile rubber tubing. The rate of flow through the entire system was controlled by a circular transfusion pump adjusted to a speed to allow each cubic centimeter of blood to be irradiated for one second. The apparatus used was the Knott Hemo-Irradiator, and the technic was that of Knott, Miley, Rebbeck and others[2] studying irradiation of the blood.[3]

On Jan. 10, 1942, 3 Macaca mulatta monkeys were each given an intracerebral injection of 0.5 cc. of a 5 per cent suspension of Flexner's M. V. strain. Symptoms of poliomyelitis developed in all the animals on January 16. We started to treat two of the animals daily, but the paralysis present was not arrested; quadriplegia occurred in both animals. One died after the second treatment; the other lived

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1. (*a*) Toomey, J. A.: Inactivation of Poliomyelitis Virus by Ultraviolet Irradiation, Am. J. Dis. Child. **54**:1490 (June) 1937. (*b*) Schultz, E. W.: Recent Advances in the Study of Poliomyelitis, J. Pediat. **1**:358, 1932.

2. Hancock, V. K., and Knott, E. K.: Irradiated Blood Transfusion in the Treatment of Infections, Northwest Med. **33**:200, 1934. Barrett, H. A.: Irradiation of Auto-Transfused Blood by Ultraviolet Spectral Energy: Results of Therapy in One Hundred and Ten Cases, M. Clin. North America **24**:723, 1940. Miley, G.: The Ultraviolet Irradiation of Autotransfused Human Blood: Studies in the Oxygen Absorption Value, Am. J. M. Sc. **197**:873, 1939; The Knott Technic of Ultraviolet Blood Irradiation in Acute Pyogenic Infections: A Study of One Hundred and Three Cases with Clinical Observations on the Effects of a New Therapeutic Agent, New York State J. Med. **42**:1, 1942. Rebbeck, E. W.: Ultraviolet Irradiation of Autotransfused Blood in the Treatment of Puerperal Sepsis, Am. J. Surg. **54**:691, 1941; Ultraviolet Irradiation of Autotransfused Blood in the Treatment of Postabortional Sepsis, ibid. **55**:476, 1942.

3. Dr. Roswell Lowry provided the irradiation instrument and gave technical assistance in preparing the irradiated blood.

long enough for a third treatment, but it too died. The control animal showed quadriplegia within a day before one and a day after the other treated animal. It died later.

CONCLUSION

Blood obtained from Macaca mulatta monkeys experimentally infected with poliomyelitis and treated with ultraviolet rays did not modify the severity of the disease.

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UNIFORMITY OF RIBOFLAVIN CONTENT OF MILK PRODUCED UNDER STANDARDIZED CONDITIONS

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Milk is generally accepted as being one of the rich natural sources of riboflavin. Since milk is readily available, it is commonly recommended as an excellent source of riboflavin for the human dietary. However, a careful inspection of the literature soon reveals that the riboflavin content of milk is extremely variable. For instance, Williams, Cheldelin and Mitchell [1] reported that Jersey milk contained as little as 0.53 mg. of riboflavin per liter, while Lindholm [2] found as much as 7.8 mg. in Jersey milk. In other words, the amount of riboflavin reported by Lindholm is nearly 1,500 per cent of that reported by the other authors even though both studies were conducted on milk produced by the same breed of dairy cows. Such data may well cause the physician to wonder concerning the riboflavin content of the milk that he recommends to his patients. Furthermore, Hand and Sharp [3] found that summer milk contains 20 per cent more riboflavin than winter milk, and Kon [4] stated that summer milk may be even 50 per cent higher in riboflavin than winter milk. Kramer, Dickman, Hildreth, Kunerth and Riddell [5] in a study of riboflavin content of milk produced by Jersey, Guernsey and Holstein cows maintained on a very dry pasture during a drought found that after subsequent rains the same pasture, which supplied green grass, enabled the cows to produce milk 25 per cent higher in riboflavin. These and other data reported in the literature raise a question as to what extent milk can be relied on as a souce of riboflavin.

This question becomes extremely important under the rationing program, when some of the other riboflavin-rich foods are less available than formerly. Hence, it has appeared highly desirable to determine the degree of uniformity of the riboflavin content of milk produced under standardized conditions, and the present study was inaugurated for that purpose.

The milk used in this study was produced by the college herd, which included five common breeds of dairy cows. They were all normal, healthy animals maintained under constant veterinary supervision. The feeding and management conditions were similar to those employed for modern dairy farms. The winter season was selected for the study so that the standardized program could be maintained for a considerable period. In order to minimize the effect of the

Contribution 480, Massachusetts Agricultural Experiment Station.

1. Williams, R. J.; Cheldelin, V. H., and Mitchell, H. K.: The B Vitamin Content of Milk from Animals of Different Species: II. Studies on the Content of Tissues, Publication 4237, University of Texas, 1942, p. 97.

2. Lindholm, H.: The Response of Rats to Graded Doses of Flavin, Biochem. J. **32**:314, 1938.

3. Hand, D. B., and Sharp, P. F.: The Riboflavin Content of Cows Milk, J. Dairy Sc. **22**:779, 1939.

4. Kon, S. K.: The Nutritional Value of Milk and Milk Products, J. Dairy Research **11**:196, 1940.

5. Kramer, M. M.; Dickman, R. M.; Hildreth, M. D.; Kunerth, B. L., and Riddell, W. H.: The Riboflavin Value of Milk, J. Dairy Sc. **22**:753, 1939.

previous pasture ration, the cows were maintained on the winter schedule for a month or more before any samples of milk were collected for analysis. The composition of the ration and the amounts fed per cow per day are reported in table 1 for each month of the experimental period.

With the exception of December, when the cows were fed mangels, the components of the ration were the same, but the amounts of the components consumed varied some from month to month. However, one may properly question what effect this had on the amount of riboflavin available to the cow, since it has been shown that cows can synthesize riboflavin in the alimentary tract. For instance, Bechdel, Honeywell, Dutcher and Knutsen [6] twelve hours after feeding removed through a permanent fistula the wet fermented feed from a heifer that had always received a ration deficient in vitamin B. They concluded from their assays that vitamin B complex was produced in the rumen of the experimental cow by bacterial fermentation with Flavobacterium vitarumen. Hunt, Burroughs, Bethke, Schalk

TABLE 1.—*Composition of the Ration (Pounds per Cow per Day)*

	December	January	February	March
Locally grown hay	8	8	6	7
Corn silage	22.5	18	18	20
Grass silage	10.5	18	15	14
Beet pulp	2	3	3.5	3
Mangels	14	..	..	..
Grain (ground) 14 per cent protein	9.7	9	9	9.5

TABLE 2.—*Stage of Lactation*

	December		January		February		March	
Breed	No. of Cows	Days, Average	No. of Cows	Days, Average	No. of Cows	Days, Average	No. of Cows	Days, Average
Ayrshire	16	113	16	102	18	120	18	142
Guernsey	16	123	15	136	13	134	14	152
Holstein	20	93	18	106	18	133	18	160
Jersey	12	110	12	127	11	142	12	158
Shorthorn	6	118	8	82	7	115	7	143
Average	..	109	..	112	..	129	..	152

and Gerlaugh [7] have presented data showing that riboflavin was synthesized in the rumen of steers fed corn, alfalfa, hay and a protein supplement, but that no synthesis of riboflavin occurred when the corn was omitted. McElroy and Goss [8] reported that a cow maintained on a riboflavin-deficient ration that furnished only 1.8 mg. of riboflavin per day produced milk which contained between 16 mg. and 18 mg. per day.

Obviously, it would have been desirable to maintain the same stage of lactation for the duration of the study, but this was not possible, even with the occasional addition of recently freshened cows to the herd and the removal of cows that had reached the end of their lactation period. Data concerning the stage of lactation

6. Bechdel, S. J.; Honeywell, H. E.; Dutcher, R. A., and Knutsen, M. H.: Synthesis of Vitamin B in the Rumen of the Cow, J. Biol. Chem. **80**:231, 1928.

7. Hunt, C. H.; Burroughs, E. W.; Bethke, R. M.; Schalk, A. F., and Gerlaugh, P.: Further Studies on Riboflavin and Thiamin in the Rumen Content of Cattle: II, J. Nutrition **25**:207, 1943.

8. McElroy, L. W., and Goss, W.: A Quantitative Study of Vitamin in the Rumen Content of Sheep and Cows Fed Vitamin-Low Diets, J. Nutrition **20**:527, 1940.

of the experimental cows as computed for the middle of each month of the experimental period is reported in table 2. The number of cows varied somewhat from month to month and the average stage of lactation varied for the different breeds and also varied from month to month. The herd average was one hundred and nine days for December, one hundred and twelve days for January, one hundred and twenty-nine days for February and one hundred and fifty-two days for March. Thus, the stage of lactation varied only forty-three days during the four month study. Consequently, it may be assumed that for all practical purposes the stage of lactation was uniform during the experimental period.

The cows were milked with milking machines, and the milk was cooled and mixed in covered equipment. Thus, contact of milk with light was slight, and the destructive effect of light on riboflavin was reduced to a minimum. The

TABLE 3.—*Riboflavin Content of Winter Milk*

December		January		February		March	
Sample No.	Riboflavin, Mg./Liter	Sample No.	Riboflavin, Mg./Liter	Sample No.	Riboflavin, Mg./Liter	Sample No.	Riboflavin, Mg./Liter
1	1.26	23	1.53	45	1.75	63	1.53
2	1.67	24	1.44	46	1.58	64	1.54
3	1.52	25	1.54	47	1.39	65	1.48
4	1.21	26	1.47	48	1.58	66	1.52
5	1.13	27	1.51	49	1.50	67	1.65
6	1.48	28	1.47	50	1.56	68	1.49
7	1.46	29	1.66	51	1.40	69	1.63
8	1.38	30	1.34	52	1.35	70	1.43
9	1.58	31	1.43	53	1.44	71	1.32
10	1.30	32	1.42	54	1.49	72	1.38
11	1.34	33	1.43	55	1.43	73	1.44
12	1.39	34	1.41	56	1.55	74	1.39
13	1.48	35	1.52	57	1.72	75	1.37
14	1.45	36	1.42	58	1.42	76	1.47
15	1.53	37	1.56	59	1.38	77	1.45
16	1.51	38	1.47	60	1.34	78	1.37
17	1.42	39	1.53	61	1.44	79	1.35
18	1.67	40	1.71	62	1.44	80	1.48
19	1.60	41	1.56				
20	1.65	42	1.52				
21	1.42	43	1.55				
22	1.39	44	1.56				
Average	1.45	Average	1.50	Average	1.49	Average	1.46

milk from the 3:30 p. m. milking was promtly cooled and held at 40 F. until the following morning. When the milk from the 3:30 a. m. milking had been cooled, the two were combined, and the 800 quarts (760 liters) of milk was thoroughly mixed. At this time (8:30 a. m.), when the combined milk was between ten and eleven hours old, a sample was taken, and the riboflavin assay was commenced within a half-hour.

Acetone was added to the milk to precipitate the casein, and the riboflavin content of the filtrate was determined by the fluorescence method with a Coleman Universal Spectrophotometer adapted for fluorescence measurements. The reader is referred to an earlier paper [9] for a detailed description of the assay procedure.

Since riboflavin is destroyed by light, amber glassware was used to exclude light and the assays were made in a laboratory from which all daylight was excluded by covering the windows with light-impervious paper. The room, which had a

9. Holmes, A. D.; Jones, C. P.; Wertz, A. W., and Kuzmeski, J. W.: The Ratio of Ascorbic Acid, Riboflavin and Thiamine in Raw and Pasteurized Milk, J. Nutrition **26**:337, 1943.

low ceiling, was lighted with 100 watt bulbs, but these were surrounded with long metal cylinders that prevented direct light from reaching the laboratory tables.

The results of the riboflavin assays of eighty lots of 800 quarts of milk are reported in table 3. Inspection of the data will reveal that during the first month of the study the riboflavin content of the milk varied from 1.13 to 1.69 mg. per liter, with an average of 1.45 mg. During January, the riboflavin content of the twenty-two samples of milk ranged from 1.34 to 1.71 mg. per liter and averaged 1.50 mg. The eighteen samples of milk assayed in the month of February showed a variation in riboflavin content of from 1.34 to 1.75 mg. per liter, with an average of 1.49 mg. In March the milk contained from 1.32 to 1.65 mg. per liter and averaged 1.46 mg. Thus, the monthly average values for the riboflavin content of the milk varied only by 0.05 mg. Considering the uniformity of the riboflavin content of the milk month by month, one will note that during December 73 per cent of the samples contained within 10 per cent of the average amount of riboflavin, 1.45 mg. per liter. During January, over 86 per cent of the samples contained within 10 per cent of the average, 1.50 mg. per liter. In February over 88 per cent and in March 94 per cent of the samples had within 10 per cent of the average riboflavin content. These results seem to justify the conclusion that it is possible to standardize the feeding and management conditions for a dairy herd sufficiently so that milk of relatively uniform riboflavin content may be produced for considerable periods of time. This would be particularly true for dairy herds that are stall fed continuously year after year. Such a source of milk would be especially helpful to the physician who desires for his patients milk with a dependable, uniform riboflavin content.

Prof. Clarence H. Parsons and Mr. Elliott Greenwood provided the samples of milk and furnished the data regarding the feeding and management of the cows.

ACQUIRED SYPHILIS IN INFANTS AND IN CHILDREN

REPORT OF THREE CASES

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Syphilis may be acquired even by extremely young children. A review of the American literature reveals surprisingly few reported cases of acquired syphilis in infants and young children. The foreign literature, especially the French, is rather rich in studies of this subject.

Downie [1] in 1894 reported 4 cases of acquired syphilis in children. A chancre appeared on the lower lip of each of the 4 children, and secondary lesions were noted in 3 of them. The source of infection was relatives and a roomer in the home. The mode of transmission of the acquired disease was family relations.

Fournier [2] in 1902 stated that a child may acquire syphilis and that it does not constitute "a rare rarity." He listed parturition, breast feeding, kissing, family relations and use of pacifiers and various types of toys as well as personal utensils as the manner of transmission of the disease to children. He illustrated this by reporting a case in which an infant acquired syphilis through a trumpet blown by a syphilitic visitor to the home.

In a report from Vienna [3] in 1924, acquired syphilis in children was said to be not as frequent as "heredosyphilis," of which there was an alarmingly high percentage of cases.

Latapi [4] has discussed cases of acquired syphilis in infants and children in Mexico.

The American authors, including Stokes,[5] Moore,[6] Jeans,[7] Towle,[8] and White,[9] have not contributed heavily to the literature on this disease acquired by infants and children.

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1. Downie, W.: An Account of Some Cases of Acquired Syphilis in Childhood, Four by Lip Chancre, Lancet **1**:1126, 1894.
2. Fournier, P.: La syphilis infantile acquise, Rev. internat. de méd. et de chir. **13**:1, 1902.
3. Venereal Diseases in Children, Foreign Letters (Vienna), J. A. M. A. **82**: 809 (March 8) 1924.
4. Latapi, F.: Sífilis adquirida en el niño, Gac. méd. de México **72**: 555, 1942.
5. Stokes, J. H.: Modern Clinical Syphilology, ed. 2, Philadelphia, W. B. Saunders Company, 1934.
6. Moore, J. E.: Modern Treatment of Syphilis, Springfield, Ill.., Charles C Thomas, Publisher, 1941.
7. Jeans, P. C.: A Review of the Literature of Syphilis in Infancy and Childhood, Am. J. Dis. Child. **20**:55 (July); 132 (Aug.) 1920.
8. Towle, H. P.: A Review of the Literature of Syphilis in Infancy and Childhood, Am. J. Dis. Child. **12**:406 (Oct.) 1916.
9. White, P. J.: A Review of the Literature of Syphilis in Infancy and Childhood, Am. J. Dis. Child. **23**:442 (May) 1922.

White [9] reported 9 cases, Waugh [10] 35 cases and Smith [11] 100 cases of acquired syphilis in infants and children.

Many of the authorities have now agreed that acquired syphilis occurs more frequently than is generally supposed. Both Downie [1] and Fournier [2] recognized the acquisition of this disease by children of tender years. Downie [1] believed the incidence to be rare, but Waugh [10] stated that the number of such cases is greater than generally recognized. Jeans [7] and Towle [8] did not relate any such cases. Moore [6] discussed the prognosis of infants with the disease, while Stokes [5] did not list any cases. However, he stated that congenital infections are confused with infections of extragenital origin contracted in childhood. He stated that Hochsinger recognized the elements in the confusion of congenital with acquired syphilis. Stokes [5] said: "Congenital syphilis may be applied more strictly to that type of the disease which follows infection of the fetus in passing through the birth canal. Congenital syphilis is thus a variety of acquired syphilis, and its course differs considerably from that of the prenatal or 'heredosyphilitic type.'"

In infants the disease may simulate to some extent congenital syphilis. Holt [12] agreed that the assumption that any infantile syphilis is congenital is erroneous. The acquired type of syphilis, except in the case of syphilis resulting from a transfusion, manifests itself by a typical primary lesion which is followed by secondary and tertiary manifestations unless it is recognized and therapy is instituted. Attention is directed to the danger of not recognizing a primary lesion in an infant with acquired syphilis. Interstitial keratitis occurs rarely in the secondary period of acquired syphilis, according to Stokes.[5]

Holt [12] listed parents and friends as the source of infection in most cases in which infants have acquired this disease. If the disease has been transmitted as the result of a transfusion, in Holt's [12] opinion the prognosis is "correspondingly good" when the condition is recognized early.

A complete study of the family should be made when syphilis is the diagnosis in the case of an infant or a young child. The number of infected persons found is increased by a thorough search.

As an addition to the literature on this subject, the following cases of acquired syphilis in infants and children are presented:

REPORT OF CASES

CASE 1.—*Acquired (secondary) syphilis.*

S. P., a Negro girl, was born May 10, 1940 and was 1 year and 11 months old at the time of the diagnosis of syphilis. There were a number of boarders in her home who were suspected of being the source of infection. They moved away without leaving a forwarding address.

Results of the mother's serologic tests had been repeatedly negative during her past pregnancies, and at no time did she have a history of syphilis or give any evidence of having this disease. She was carefully checked by physicians of the Maternal and Child Welfare Clinic of the Health Department of the District of Columbia. All of the other children of this mother were also carefully checked and examined, and they were found free of syphilitic infection.

S. P. was brought to the Pediatric Clinic of Children's Hospital because of fever and general malaise. Diagnosis was not made at the clinic, but the patient was hospitalized. There it was found that she had secondary syphilis. Typical condylomata lata were recognized. A test of her blood serum gave a positive reaction. Antisyphilitic therapy was given from Feb. 7,

10. Waugh, J. R.: Acquired Syphilis in Infancy and Childhood: Report of Thirty-Five Cases, Am. J. Syph., Gonor. & Ven. Dis. **22**:607, 1938.

11. Smith, R. F.: Acquired Syphilis in Children, Am. J. Syph., Gonor. & Ven. Dis. **23**: 165, 1939.

12. Holt, L. E., Jr., and McIntosh, R.: Diseases of Infancy and Childhood, New York, D. Appleton-Century Company, Inc., 1940, pp. 1238-1239.

1942 to February 28, at which time she was discharged from that hospital. About two months elapsed before S. P. was brought by her mother to the Pediatric Clinic in the Maternal and Child Welfare Clinic of the Health Department of the District of Columbia, although immediate referral had been made. On her first visit a physical examination did not reveal any symptoms of secondary syphilis; so a diagnosis of early latent syphilis was made, and therapy was instituted.

After the first visit the child failed to return. She was brought back for treatment on July 6, 1942 and has continued to receive therapy regularly. Later, with the résumé of findings from the Children's Hospital and the mother's history of negative reactions to serologic tests before and during each of her various pregnancies, the original diagnosis of early latent syphilis was changed to that of secondary syphilis, which conformed to the diagnosis made at the time of hospitalization of the patient.

CASE 2.—*Acquired (secondary) syphilis.*

B. G., a boy, born Aug. 29, 1940 and aged 1 year and 10 months at time of the diagnosis of syphilis, had been infected by a sexually delinquent aunt, I. K. G., who had a highly infectious type of this disease.

B. G. had been under the care of the well baby clinic of the Maternal and Child Welfare Clinic of the Health Department of the District of Columbia, from the time he was 2 months old. He had an uneventful early life. When 1 year and 10 months old, he had pains in his legs and arms and was examined at Gallinger Municipal Hospital. He was referred to the venereal disease clinic of the health department for therapy. Results of Kahn's serologic test showed a 4 plus reaction, and roentgen examination of the long bones revealed periostitis. A diagnosis of syphilitic periostitis was considered. On further examination the child showed a fading secondary rash on the scrotum and enlarged cervical and inguinal glands. The right arm at the elbow joint was swollen and was tender to touch; movements, both passive and active, were considerably restricted. There was pain in all extremities, of maximum intensity in the right arm. Reactions to repeated serologic tests proved to be positive. The diagnosis of secondary syphilis was made, and the child was put under antisyphilitic therapy. After one injection of sulfarsphenamine the rash on the scrotum disappeared and the swelling of the right arm diminished, tenderness being greatly reduced.

The mother of B. G. had been under the supervision of the Maternal and Child Welfare Clinic since Oct. 7, 1940; she had continued to have negative reactions to serologic tests during her several pregnancies and was reported as free from syphilis.

In an examination of a 4 year old sister of B. G., the blood gave a negative reaction on July 8, 1942.

A diagnosis of congenital syphilis can be safely excluded, as the mother definitely has never had and does not now have syphilis. Further inquiry showed an aunt to be the source of infection of B. G.

The aunt, I. K. G., attended the venereal disease clinic of the health department, after a report from the Department of Health of North Carolina stated that she was "a highly infectious syphilitic without therapy." Her infection was diagnosed as secondary syphilis. She was involved in the first test case of its kind in the courts of the District of Columbia, where legal measures for isolation had to be taken to protect the community from persons infected with syphilis.

CASE 3.—*Acquired (early latent) syphilis.*

E. G., next older brother of B. G. (case 2), was infected by his aunt, I. K. G., who had a stubborn and extremely infectious form of syphilis.

E. G. had a positive serologic reaction of the blood. No history of primary or secondary manifestations was revealed. A diagnosis of early latent syphilis was made. Tests of the mother's blood gave negative results before and during each pregnancy. Any possibility of the presence of congenital syphilis was ruled out in this case. The same syphilitic aunt, I. K. G., was the source of infection.

COMMENT

It is our belief that primary lesions are frequently unrecognized in infants with acquired syphilis. The disease in one member of siblings should arouse a suspicion of acquired syphilis if there is no evidence of the mother's having or having had syphilis. Since the risk of subsequent development of syphilis of the cardiovascular or the central nervous system is greater in infants with acquired syphilis than in those with congenital syphilis (Moore [6]), emphasis must be placed on the importance of alertness to the discovery of this type of disease.

Serologic tests and roentgenograms should be used when symptoms are absent. All lesions should be carefully noted and followed systematically by a Wassermann test as a means of prevention of subsequent, more serious difficulties. Infants in whom the disease is diagnosed early have a more favorable prognosis as to "cure." Complete studies of the families should be made in all such cases to insure prompt location of infected persons and early therapy.

The number of cases of acquired syphilis in children is increased largely by social, moral and economic conditions in the community. Overcrowding of rooms, lack of adequate sanitation and poor economic conditions with accompanying lowered moral standards play a definite part in the spread of this type of disease.

Prevention of new instances of acquired syphilis in children will result from the amelioration of these contributing factors and from the improvement of social and economic conditions in each community. Training physicians to recognize early this acquired type of syphilis and to administer antisyphilitic therapy immediately will prevent later tragedy.

Dr. George C. Ruhland, Health Officer, District of Columbia, and members of his staff cooperated in this study.

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FOLDS AND CREASES OF THE SKIN DURING CHILDHOOD

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There is hardly anything to be found in the pediatric literature on the subject of the folds and creases of the skin, their appearance and disappearance, their character and their relations to other changes of the body. This omission is striking, since these folds and creases belong among the peculiarities which make the body of the infant and the child appear different from the body of the adult and which, on the other hand, have always been known to laymen and physicians, to painters and sculptors. But they seem to have escaped the attention of physicians, and it is only occasionally that observations are encountered concerning them in descriptions or characterizations of patients. Since these features may contribute to the diagnosis of morbid conditions and to the differentiation of stages of development, they may also contain some value for the practicing physician, especially the pediatrician.

The general content of this article is the discussion of the following topics: (1) the big creases of the body of the infant; (2) the development of the creases of the elbow, and (3) the big flexor creases of the palm.

THE BIG CREASES OF THE BODY OF THE INFANT

The big creases are most striking on the thighs of the young child. But analogous formations are also to be found on the upper and lower parts of the arms, on the trunk (groin and axillary region) and on the face of the newly born infant, and during the first three months of life they are encountered on the abdomen and on the back, where they are most distinct in the flexor position of the parts in question (bending of the trunk to one side, etc.).

The number and intensity of the folds are extremely variable on all parts of the body; their variability is so great that it would hardly be possible to find two children with identical formation of creases. This holds true as well for location and number as for length and depth of the furrows. In addition to the large creases on the thighs, of which there are generally three, usually on the inner aspects, there are also a variable number of shallower creases in the region of the knees and ankles. At the same time one finds ordinarily one or two deep furrows on the upper part of the arms, above the elbow creases, and frequently one deeply notched furrow on the lower part of the arms.

The pattern of the creases of the trunk is variable too; sometimes they are scanty and sometimes numerous. Regularly two or three creases of medium depth are to be found on the back in the region of the sacral bone. Almost all young children have also one to two deep transverse creases on the lower part of the abdomen. As the trunk is bent sideward, creases appear parallel to the ribs from the axilla, extending sometimes to the pelvic bone, in all young children. The pattern of the creases is often simple, but on other infants extraordinarily complicated and striking patterns appear; e. g., eight to ten deep parallel creases in certain postures.

The creases of the right and left sides of the body are never fully symmetric; often they are of striking asymmetry as to number, arrangement and depth. Neither do the creases of the right leg correspond to those of the left. It is not justifiable, however, to draw any conclusions from these deviations. For instance, one cannot infer from them asymmetries or one-sided malformations of deep soft tissues or bones, for example the presence of a congenital luxation. In a healthy child only the creases corresponding to the articular spaces of the knee and elbow, the so-called Langer lines, are symmetric. When these are asymmetric (differ in level), one may suspect a disturbance in the anlage of the deeper tissues.

At the end of the first year or in the course of the second year all these creases disappear. The disappearance progresses surprisingly fast. In only a few weeks even deep folds become leveled, without a simultaneous change in the intensity of the cushion of fat (panniculus adiposus).

The opinion has prevailed that it is the development of the sense of equilibrium and especially changes in coordination and tonus of the muscles which have some connection with the disappearance of the folds. That assumption implies a role of influences referable to the central nervous system in the formation and fading away of the creases. Therefore children with delayed development of equilibrium or children with diseases of the central nervous system retain the pattern of the folds of early life longer. In such children the creases generally specific for the body of the infant remain until the third year of life or even later. Thus I have known of the persistence of all the creases of the body in a child with congenital myatonia. A second child, with encephalitis in his first year of life which left him with a remnant of mental debility but without other motor lesions, showed distinct creases of the abdomen and arms even at the end of the fourth year of life. A third child, who had Feer's neurosis (acrodynia) in his second year of life, retained remnants of the creases up to his fifth year.

At the end of the second year of life or at the latest in the third year in all healthy children only the creases in the groin and in the bends of the big joints are present, and these remain on the body of the older child and of the adult.

The significance of the creases of the skin and their development have neither been studied nor explained. Of older writers only Michael Cohn reported in detail on the creases of the thigh of the child. Fischl described peculiar, quickly vanishing creases at the root of the nose and around the eyes of newly born infants and of infants during the first three months of life.

A series of theories have been ventured for the development of the creases. According to the opinion of a few anatomists, they are produced by the pushing together of the skin by the panniculus adiposus. Other writers explained their development as a consequence of the posture of the child in utero. In favor of this view was the presumption that the creases are more distinct in bent limbs than in outstretched limbs, a point of view not in accord with the facts. Froriep referred to them as "drapery folds," since they resemble the folds which appear in drapes when they are closed and opened.

There are important objections to be made against each of these views:

1. The formation of the creases is fully independent of the amount of the panniculus. Children born prematurely, as early as the seventh to eighth fetal month, show the formations distinctly, although they are born without any panniculus. Neither do the creases disappear in an infant emaciated by disease; in fact, they remain even after the loss of all the cushions of fat. On the contrary, the wrinkling of the skin due to emaciation augments the creases in atrophic infants. But these wrinkles of emaciation are readily distinguished from the

creases specific for the body of the infant during sickness and health. On the other hand, there is no connection between the amount of the panniculus of an infant and the intensity of the creases. The amount of increase of fat within the first year and the absolute or relative physiologic decrease of fat of the second and third years of life have therefore no bearing on the shorter or longer presence of the creases. Fat children and lean children show this formation of creases in the same way, regardless of the stronger or weaker development of the panniculus. Atrophic infants retain the creases during the period of emaciation, and the folds remain unchanged as to localization and size when the atrophy heals, with the deposit of larger amounts of fat. Size, depth and localization of the creases represent, rather, individual characteristics of each child. Fat children not rarely show few and indistinct creases; lean children often show pronounced folds. The creases disappear in the majority of the children in the course of the second year of life independent of the intensity of the panniculus. In later years or in the adult stage even the heaviest development of fat does not cause the re-formation of creases typical of early childhood.

2. The posture of the fetus in utero as affecting the origin of the creases seems unimportant. The prematurely born child with less body length, which is carried in utero hardly flexed, does not show any variation in the formation of creases from that of the infant born with normal body length and higher birth weight.

3. The extension of the legs does not make the creases disappear, nor does the flexion of the legs cause an essential increase of the creases. The creases located between the joints preserve approximately equal depth and shape in almost all postures.

4. A comparison of the creases to drapery folds does not agree with the facts. Such folds, which come about by the streching or the pushing together of a material, run in the direction of the strain when stretched and in a direction perpendicular to this when released. Neither of these corresponds to the direction of the folds of the child. Here the creases run independently of strain and release obliquely to the supposed or theoretically demanded direction of the traction. Moreover, it is dubious whether the amount of traction (strain) or release would be enough to explain the sometimes enormous intensity of the creases.

In view of the lack of a definite explanation of the appearance and disappearance of these crease formations it seemed to be desirable to look for their anatomic basis. The histologic examination of excised creases of skin of stillborn children did not yield any conclusive information, according to the judgment of Dr. Caspar and Prof. Koenigstein. The structure of the skin did not show any changes which would explain the origin of the creases on just these spots. Thus the histologic picture does not produce the explanation looked for.

Since anatomic findings cannot explain the production of creases, it is obvious to think of functional peculiarities of the skin specific to the age. There already are sufficient foundations for such a presumption. Examinations of the skin of young children by Weiss [1] showed that the skin undergoes essential changes for a long time after birth, changes especially of a quantitative and structural kind. The skin apparently belongs to those organs with a "function in the making" (werdende Funktion), as Salge called them. These changes concern the amount and structure of the elastic tissue and, above all, the structure of the connective tissue. In a young child these tissues are still immature, unarranged and sparsely

1. Weiss, F.: Jahrb. f. Kinderh. **135**:272, 1932.

developed. Accordingly, Wilson [2] found as a substrate for the fine wrinkles peculiarities of the fibrous and elastic structure of the tissue. The tissue assumes only slowly the structure of that in the adult body. These changes recall to a certain degree the formation of the fine osseous structures as they are arranged under the influence of mechanical requirements. It seems not improbable that the connective and elastic tissue of the skin too gets shaped only slowly under the stimulus of functional requirement. An external expression of this maturing process may be perhaps the disappearance of the creases, as it takes place in the course of the second year, that is, during the time of the development of mechanical functions. If there is a delay in the development of these functions or a lack of stimuli from the central nervous system which have a determinating influence in the course of the process or if the nervous centers whose task it is to develop the mechanical functions are diseased, the pattern of the creases is retained beyond the normal time. Hence it is readily understood why, as mentioned previously, children with myatonia or young children with healed encephalitis retain the creases specific for infancy beyond the normal time.

THE FOLDS OF THE ELBOW

The biologic conditioning and the relationship of the creases of the skin of man are demonstrated especially well by the development of the creases of the elbow, the crease formations to be found at the outside of the arm in the region of the olecranon. These creases usually show the following development, conditioned by age and sex. (The inspection of this region is done best with the arm slightly bent, at an angle of about 160 degrees; extreme extension causes folds of a different sort.) In the newborn child and in the infant during the first year of life there are one to two dimples or short creases which run straight or obliquely across the arm, the depth and length of which fluctuate within not too broad limits in each individual child. These creases are not dependent on the development of the panniculus. They are of the same form on a newborn baby, with relatively scanty or missing panniculus, as on a well nourished or fattened infant. They are retained during extreme emaciation in the same way as are the creases of the thighs or of other parts of the body. And the lean child born prematurely during the seventh or eighth month of pregnancy shows also the peculiarities of relief of the surface, although sometimes on him they are less pronounced. Independent of the further formation of the panniculus, these folds remain in the same shape until about the third year of life. In the majority of children these folds disappear relatively quickly at this age, and from about the third to the sixth year of life the region of the elbow appears to become smooth and without any "wrinkle relief;" it is only when the arm is illuminated from the side that the olecranon and the bellies of the muscles appear as dainty shadows. This smooth elbow is found unchanged in boys and girls during the entire age of youth. During the third decade men and women show the same pattern of relief of the elbow. But at the end of the third decade or more frequently at the beginning of the fourth decade a striking differentiation of the sexes begins to take place in respect to the formation of the creases in this region (fig. 1). While in men there occur no creases at all in the skin of the elbow or there is just a moderate and belated one up to old age, there appear here on the woman of 30 years cross folds which increase in number and intensity with the increasing number of years. These creases appear on every woman, as a rule, regardless of whether other signs of premature aging or maturity come about or whether the beginning of the fourth decade

2. Wilson, E.: Lancet **1**:422, 1941.

has been hidden by the retention of a naturally youthful appearance or by good care. These formations peculiar to women have certainly no relation to the amount of the panniculus. The slender woman shows her age-telling creases in the same manner as the stout woman does. These age-conditioned and sex-conditioned creases have nothing in common with the wrinkles and dimples which render the elbow of the young child so attractive. They are new formations of a peculiar type appearing in women at the age of 30 years, while men, but for rare exceptions, escape this symptom of approaching age.

From a broader point of view, the recording of such courses and changes appears to be instructive, because they show that even the smallest peculiarities in the structure of the body are not the results of coincidence. Each change in the body follows its own biologic laws, which may be apprehended on the basis of such experiences. After all, the summation of such details marks the peculiarities in the structure of the individual organism, and the basic facts for a more exact comprehension of the constitution of the individual result from a totality of such observations.

Fig. 1.—Creases near the elbow: *A*, a 48 year old woman; *B*, a 30 year old woman; *C*, a 38 year old woman; *D*, a 55 year old man.

THE FLEXOR CREASES OF THE PALM

At the present time the lines of the human palm are interpreted by anatomists as a system of flexion and kinking furrows in the leotard-like cutaneous vestment. Accordingly the lines of the palm are to a certain extent analogous with the divisions between the plates of the epidermis armor of some animals. This mere mechanical explanation of the development of creases seems to be as little true as the attempted explanation of the big folds of the body of the child by similar conceptions. I hope to be able to demonstrate that there is a central principle involved in the formation or the lack of the large lines of the palm and in their arrangement or disarrangement.

From birth on there are the following six large flexion furrows in the human palm: (1) furrow of the thumb (great opposition furrow); (2) five finger furrow; (3) three finger furrow; (4) middlefinger furrow; (5) ringfinger furrow, and (6) small finger furrow. The expression five finger or three finger furrow indicates that the furrows run below (or at the thumb, above) five and three fingers respectively. Figure 2 represents the palm as it is found in the majority

of normal persons of Europe, America and northern and middle Asia in infinite varieties. The Mongolian race seems to have an exceptional pattern. In a certain group of morbid conditions the lines of the palm appear to be changed.

In a child with mongolism (figs. 3 and 4) the pattern of the palm is characteristic. In place of the usual three large lines, only two are to be found. This change of the picture of the lines of the palm in children with mongolism has

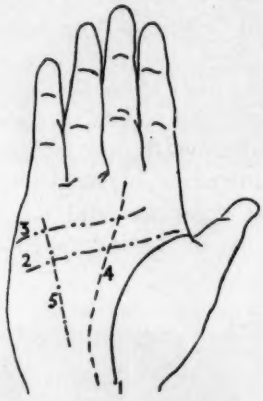

Fig. 2.—Principal lines of a normal human palm.

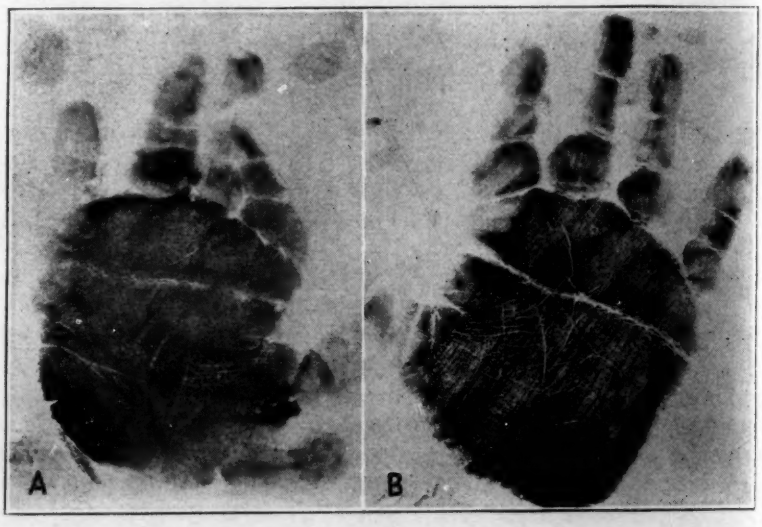

Fig. 3.—Prints of the hands of a 15 month old child with mongolism: *A*, left hand; *B*, right hand.

already been described occasionally.[3] There is found only one transverse line also in a variant form of mongolism, the *typus amstelodamensis* described by Cornelia de Lange.[4] This type is marked by hypoplasia of the whole body, hirsutism (extreme hairiness), nostrils which are pointed frontally similar to a monkey's nose, contraction of the elbow joint and flabby skin and syndactyly. In patients with mongolism

3. Doxiades, L., and Portius, W.: Ztschr. f. menschl. Vererb.- u. Konstitutionslehre **21**:384, 1937.

4. de Lange, C.: Arch. de méd. enf. **41**:193-1938.

this variety of lines of the palm is absolutely according to biologic law; hence the presence of a complete set of lines in the palm of a child otherwise of the mongoloid type should raise the suspicion of the presence of another disease.

This is illustrated by the following example:

Louis V., 12 years old and normally born, showed a delayed development by the end of the first year. He presented fully the mongoloid type (obliqueness of the eyes, epicanthus, highly roofed palate, crooked small finger and imbecility of high degree), but the palms showed normal lines. Intensive questioning of the mother revealed that the child had had a positive Wassermann reaction and that he had been given only a short course of bismuth therapy. The Wassermann reaction was still positive at 12 years. The child was congenitally syphilitic and only belatedly and insufficiently treated; in him the syphilitic infection stigmatized the organism in a fashion assuming the somatic and psychic trend of mongoloid idiocy. The lack of a defect in the lines of the palm suggested the rejection of the diagnosis of mongolism and caused a search for another reason for the peculiar body structure of the patient.

The pattern of the lines of the palm specific for patients with mongolism is regularly found in the palms of some primates, particularly the great anthropoid

Fig. 4.—Prints of the hands of a 6 month old child with mongolism. In addition, the child presented macroglossia, epicanthus, navel hernia, hypotonia and crooked small fingers.

apes, which are the only representatives among animals showing lines in the palm at all. I reject as unjustified the presumption of interpreting the lines of the palm of a person with mongolism as a relapse into or a remaining in a primitive stage of development. I shall refer later to the probable reasons in an attempt to explain this peculiarity.

COMMENT

Bagg and Little[5] succeeded in producing a recessive mutation in mice by roentgen irradiation; this mutation manifested itself by anomalies of the eyes and deformities of the foot in the descendants of the irradiated animals. The eyelid space of the newborn mice was narrowed more or less by malformation of the lids. At the extremities concretions of all the toes appeared; sometimes disturbances of development or mutilations of the skeleton of the foot were brought about.

Bonnevie was able to show in serial examinations of embryos of 7 or 8 mm. in length that a much greater amount of cerebrospinal fluid left the liquor space

5. Bagg and Little, cited by Engel.[6]

through the foramen Weedi than was the case in normal embryos. Weed's foramen is an attenuation of the layer of cells in a region of the brain anlage, which later forms the roof of the fourth ventricle. A certain amount of fluid has to leave through this foramen in order to bring about the subarachnoidal spaces. This embryonic formation may rather be compared with Magendie's foramen in respect to its function. If there is a surplus exit of fluid, it gathers in the form of blebs in the subcutis. These blebs wander about because of the state of tension of the skin and the formation of the surface of the embryo until they become arrested at places especially depressed, for instance at the eyelids or at the ends of the extremities. Here transudations and extravasations of blood increase the content of the blebs. These disturbances of development are brought about through the pressure of the blebs at the places where they finally settle in their migrations. Certain anomalies and disturbances of development in human beings are produced probably in similar fashion. Weed's foramen appears in a 7 to 8 mm. long human embryo, corresponding to a fetal age of 5 to 6 weeks, exactly above the anlages of the nuclei of the facial and abducens nerves at the roof of the rhomboid fossa. If the bleb settles at this spot, disturbances of the development of the nucleus of the facialis or of the abducens nerve may be originated by pressure effect. If the bubble leaves through Weed's foramen, the formation of a pterygium is brought about or abnormal creases are produced in the region of the neck. If the bleb continues its wandering, defects of the pectoralis muscle or hypoplasia of the mamillas, web formations or dystrophies of the nails are caused and perhaps also disturbances of the development of the pattern of the palm are possible. If several blebs migrate, multiple malformations may occur in the same manner. If the blebs spread flatly under the skin, they are the cause of so-called congenital lymphangiectatic edema, which may be found in the nape and advancing from there to more or less large parts of the trunk and to the extremities.

Concerning dyostosis multiplex (Pfaundler-Husler), its origin through migration of blebs has been made probable by Engel.[6] Dyostosis multiplex [7] is a generalized disturbance of the development of the skeleton marked by oxycephaly, scaphocephaly, hydrocephaly, opaqueness of the cornea, deep-set ears, enlargement of the hypophysis and other endocrine disturbances, dwarfism, thickenings of the skin, idiocy, multiple hernias, crura valga, pedes plani, inhibition of movement of the joints, Sprengel's deformation, cutis laxa, etc. It is presumed from the experiments of Bonnevie that a gene is injured by roentgen irradiation. This impairment causes the disturbance in the closure of the canalis cerebralis and the subdural spaces, which, in turn, causes the extravasation of an abnormally large amount of fluid. It is also assumed for the human embryo that impairment or faulty anlage of a main gene or, more probably, mechanical factors with an intrauterine effect may cause the same mutation in the earliest phases of development as was obtained arbitrarily in the animal experiments by roentgen irradiation of the genes.

In patients with malformations or deformations which are known as hereditary diseases in a stricter sense, as, for instance, the majority of congenital luxations of a hip, a normal pattern of the palm is found. This fact speaks for the assumption that in the human fetus an impairment of the primary germ anlage, the inheritance of chromosomes of the parents, cannot be considered responsible for the

6. Engel, D.: Etiology of Multiple Deformities, Am. J. Dis. Child. **60**:562 (Sept.) 1940.

7. Husler, J., in Pfaundler, M., and Schlossman, A.: The Diseases of Children, Philadelphia, J. B. Lippincott Company, 1935, vol. 2, p. 45.

production of the disturbances of development which result in the occurrence of an abnormal pattern in the lines of the palm.

SUMMARY AND CONCLUSIONS

The pattern of the lines of the palm probably furnishes a clue to the solution of the problem whether a disturbance of development is hereditary or whether it has been brought about by some influences which interfered with the regular development of the fetus after conception had taken place. An abnormal pattern of the palm as an accompaniment of a congenital disease probably indicates a disturbance of development which occurred in utero; a normal pattern of the lines of the hand suggests that from a defect or deficiency in the parental germ cells a disturbance of evolution occurred. The pattern of the palm is involved only if the disease itself, or at least its anlage, can be traced back to an early stage of intrauterine development. The pattern of the hand is fixed in utero and is found fully developed in the sixth month of pregnancy; it is not subject to any change through diseases occurring during the later months of pregnancy or soon after birth or even in the later years of childhood. Furthermore, it is to be noted that all the changes are found in the majority of the patients more distinctly in the left hand, sometimes exclusively in the left hand.

At first glance, the observations presented here concerning the occurrence of anomalies of the large lines of the palm in generalized disturbances of development may seem strange. Their occurrence, however, is after all, no more miraculous, no easier to explain and no more readily understood than the appearance of an epicanthus or a crooked small finger in a child with mongolism or the characteristic malformations of the hands and feet of patients with arachnodactyly. The collecting of more evidence seems to be of interest. It may be possible in this way to add to the desirably sharper characterization of congenital morbid conditions or to the diagnosis of many constitutional anomalies.

Case Reports

FAMILIAL CEREBRAL DEGENERATION WITH CORTICAL ATROPHY

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In the practice of pediatrics many children are examined because of backwardness of development. A number of well recognized clinical types are presented, such as mongolism, microcephaly and oxycephaly. For most of these unfortunate children one can do little therapeutically. One may, however, help the parents by giving them as accurate prognoses as possible. Many of the abnormalities are not familial, and the chance of their recurrence is extremely small. Some types, however, do occur more than once in the same family and generation. This is true of progressive subcortical encephalopathy (Schilder's disease), of amaurotic idiocy, of diffuse infantile cerebral sclerosis as described by Krabbe [1] and of a slightly different form reported by Pelizaeus [2] and by Merzbacher.[3] Penfield and Geyelin [4] described a familial cerebral calcification with epilepsy. Hereditary chronic progressive chorea (Huntington's) and the spinal form of hereditary sclerosis (Friedreich's ataxia) are well known examples of heredofamilial diseases of the nervous system, and there are numerous other less well defined conditions which are undoubtedly familial.

REPORT OF CASES

I wish to report 2 cases in which the patients, a brother and sister, apparently suffered from the same gross disorder of the brain.

CASE 1.—A girl aged 6 months was brought to the Mayo Clinic on July 19, 1941. She was the second child of healthy parents. The older child was well. Convulsive attacks had begun when the child was 5 months old. At first these consisted of "spasms" or "rigid attacks" which occurred during feedings and lasted for a few seconds only. After three days the attacks became more severe, and there was twitching of the arms and legs. Then generalized convulsions occurred five to six times daily, associated with feeding but not accompanied by fever. It was also noted that the child was taking progressively less interest in her surroundings.

When the child was brought to the clinic she weighed 15 pounds and 8 ounces (7 Kg.). Her appearance was that of a normal baby, and no spasticity was present. She did not follow a light or respond to jingling keys, did not grasp at an object when it was put in her hand, was unable to sit up and made various uncontrolled movements. Her head was not unusual in size or shape.

The value for hemoglobin was 12.9 Gm. per hundred cubic centimeters of blood, and the leukocyte count was 11,800. A flocculation test for syphilis gave a negative result, and a roentgenogram of the thorax did not disclose any abnormality. The spinal fluid was clear; it contained 30 mg. of protein per hundred cubic centimeters and 2 lymphocytes per cubic millimeter. The results of Kolmer and Kline tests were negative. An encephalogram, made after withdrawal of 65 cc. of spinal fluid, showed diffuse cortical atrophy (fig. 1).

* Section on Pediatrics, the Mayo Clinic.

1. Krabbe, K.: A New, Familial, Infantile Form of Diffuse Brain-Sclerosis, Brain **39**: 74-114 (June) 1916.

2. Pelizaeus, F.: Ueber eine eigentümliche Form spastischer Lähmung mit Cerebralerscheinungen auf hereditärer Grundlage (Multiple Sklerose), Arch. f. Psychiat. **16**:698-710, 1885.

3. Merzbacher, L.: Eine eigenartige familiär-hereditäre Erkrankungsform, Ztschr. f. d. ges. Neurol. u. Psychiat. **3**:1-138, 1910.

4. Geyelin, H. R., and Penfield, W.: Cerebral Calcification Epilepsy, Arch. Neurol. & Psychiat. **21**:1020-1043 (May) 1929.

A diagnosis of diffuse cortical atrophy with progressive mental retardation and secondary convulsive disorder was made. In July 1943 the child was still under custodial care at a vocational hospital.

CASE 2.—A boy aged 9 months, the brother of the patient in case 1, was brought to the clinic June 14, 1943, because his mother had noted that he was "backward" at the age of 5 months. She had been particularly observant because of the difficulty with the patient's sister. It had been noted that the baby paid little attention to his surroundings, did not hold or play with simple toys and was unusually quiet. The retardation apparently had been progressive. At first he was attentive to music and to other sounds; later he paid no attention to any such stimuli.

The boy weighed 18 pounds 7½ ounces (8.4 Kg.) and appeared well nourished. He was unable to sit up alone, though he held his head up feebly. He paid no attention to the examiner and made no response to a strong light or to a loud clapping noise, but he rolled his

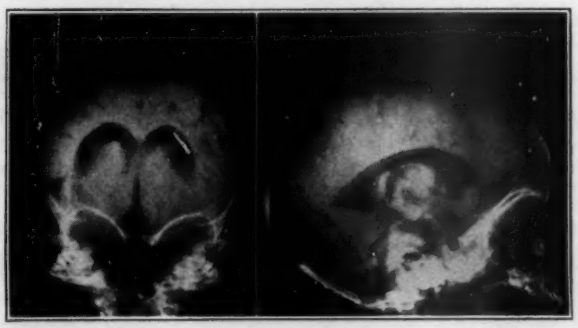

Fig. 1 (case 1).—Encephalograms, made after withdrawal of 65 cc. of spinal fluid showing diffuse cortical atrophy.

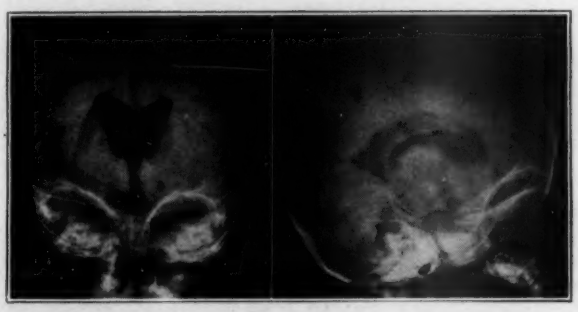

Fig. 2 (case 2).—Encephalograms, made after the withdrawal of 75 cc. of spinal fluid, showing slight ventricular enlargement and diffuse cortical atrophy, more evident on the right.

head from side to side and frequently emitted a sharp cry. There was no generalized spasticity, but the deep reflexes were increased and he tended to assume the opisthotonos position.

Urinalysis did not reveal any abnormality. The value for hemoglobin was 13 Gm. per hundred cubic centimeters of blood. The erythrocyte count was 4,180,000, and the leukocyte count 9,500. A differential leukocyte count revealed 72 per cent lymphocytes, 2 per cent monocytes, 24 per cent neutrophils and 2 per cent eosinophils. The result of a flocculation test was negative, and roentgenograms of the head and thorax were normal. A roentgenogram of the wrist showed that ossification was considerably advanced. A Mantoux test (first strength tuberculin) gave negative result. The spinal fluid was clear and under normal pressure; it contained 20 mg. of protein per hundred cubic centimeters and 4 lymphocytes per cubic millimeter. Kolmer and Kline tests on the spinal fluid gave negative results. An encephalogram, made after the withdrawal of 75 cc. of spinal fluid, showed slight ventricular enlargement and diffuse cortical atrophy (fig. 2). The latter was more evident on the right side (probably owing to incomplete drainage).

A diagnosis of diffuse cortical atrophy with progressive mental retardation was made.

COMMENT

There are three fairly well recognized conditions, namely, progressive subcortical encephalopathy (Schilder's disease), Krabbe's diffuse infantile familial cerebral sclerosis, and amaurotic familial idiocy, which must be distinguished clinically from the condition present in these 2 cases. In progressive subcortical encephalopathy, local disturbances of motility, such as hemiplegia, monoplegia and paraplegia, develop and there finally is decerebrate rigidity. Cortical blindness has been described as a promiment feature and signs of increased intracranial pressure may be present. Finally, at necropsy, swelling of the brain may be observed. Cortical atrophy is not prominent; the lesions are mainly in the white matter. Krabbe's diffuse infantile cerebral sclerosis begins at the age of 4 to 9 months with a state of excitement and, possibly, fever. This condition is followed by apathy, gradually increasing muscular rigidity and convulsions. After a few months death results from pronounced debility. Pathologically there is sclerosis of the white matter, with the cortex relatively intact. Amaurotic familial idiocy is distinguished by the appearance of the cherry red spot in the retina. At necropsy the cerebrum usually shows no gross change or malformation. Not all of these conditions are well defined, however, and in the 2 cases reported the pathologic condition cannot be finally classified until examination of brain tissue is possible.

These 2 cases show: (*a*) that, as Crothers, Vogt and Eley [5] pointed out, gross cortical lesions can be demonstrated readily by air encephalography, and (*b*) that cortical atrophy may occur more than once in any one family. There seems to be no doubt that in these two cases the changes in the encephalograms are indicative of gross changes in the brain, on which the clinical signs and symptoms depend. That this is not always so has been our experience at the clinic; it has also been pointed out by Ahnsjö,[6] who stated that gross mental defects can occur without demonstrable changes in the encephalogram and, conversely, that children with normal intelligence quotients may have abnormal encephalograms. Brines and Lord [7] also stated that the encephalogram alone is an inadequate tool for prognosis.

There is no treatment to offer save custodial supervision, and these 2 children are in a special hospital for permanent institutional care.

Mayo Clinic.

5. Crothers, B.; Vogt, E. C., and Eley, R. C.: Encephalography in Cases with Fixed Lesions of the Brain, Am. J. Dis. Child. **40**:227-246 (Aug.) 1930.

6. Ahnsjö, in discussion on Ström, J.: Några fall belysande encephalografiens diagnostiska värde i späd- och småbarnsåldern, Nord. med. **2**:1935-1940 (June 24) 1939.

7. Brines, J. K., and Lord, E.: The Place of Encephalography in Prognosis in Childhood, J. Pediat. **15**:836-843 (Dec.) 1939.

PROLAPSUS UTERI ASSOCIATED WITH SPINA BIFIDA AND CLUBFOOT IN NEWBORN INFANTS

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The literature has been briefly reviewed and a case reported previously.[1] In that case a white child died from aspiration pneumonia and infection of a spina bifida tumor on the twenty-seventh day of illness.

REPORT OF A CASE

In this case, the second of this condition observed in the University Hospital Clinic, the patient was a newborn Negro girl, whose weight was 5 pounds, 15 ounces (2,310 Gm.). She was delivered spontaneously, and she cried spontaneously. She was the fourth child in the family; all the children were born at term, but only one was still living. No history of any abnormalities in any of these was obtained. The mother was 24 years old. The placenta was discoid and apparently normal. One coil of the umbilical cord was around the child's neck at birth.

Fig. 1.—Infant with spina bifida tumor, prolapsed cervix and clubfoot.

The essential pathologic process was well illustrated and consisted mainly of the meningocele with spina bifida in the lumbosacral region, which appeared as a large soft mass, 2½ by 2 by 1¾ inches (6.3 by 5 by 4.4 cm.). This granulated and became infected, so that it was covered by a necrotic discharging membrane. When the child cried the mass became more turgid. The child's head was not abnormally large, having a circumference of 13½ inches (34 cm.). The anterior fontanel measured 3¼ inches (8.2 cm.) in length and 1½ inches (3.8 cm.) in width. The sutures were open, and the anterior fontanel connected with the posterior fontanel. The external genitals were normal except for a red granular-appearing cervix protruding between the labia majora to an extent of a centimeter or more when the child cried. A ring of rectal mucosa projected from the anus. The left foot was internally rotated and had extreme talipes varus. The same pathologic condition appeared in the right foot but not to such a degree.

Results of laboratory studies of the child were as follows: hemoglobin content, 10.3 Gm.; red blood cells, 2,330,000; white blood cells, 10,300; lymphocytes, 90 per cent; polymorphonuclear cells, 6 per cent, and monocytes, 4 per cent; Wassermann and Kahn reactions, negative. A specimen of urine was not available.

The weight curve went down several ounces and then remained stationary. The temperature gradually rose daily to the time of death, on the eleventh day, when it was 103 F.

From the Department of Obstetrics and Gynecology and the Department of Pediatrics, University of Georgia School of Medicine.

1. Torpin, R.: Prolapsus Uteri Associated with Spina Bifida and Clubfeet in Newborn Infants, Am. J. Obst. & Gynec. **43**:892, 1942.

Apparently the meningocele tumor had ruptured prior to that time, and this may have been an immediate factor.

These rare but tragic and interesting cases, of which there are upward of 40 in the literature, direct attention to the important role of the nerve supply to any outlying portion of the body.

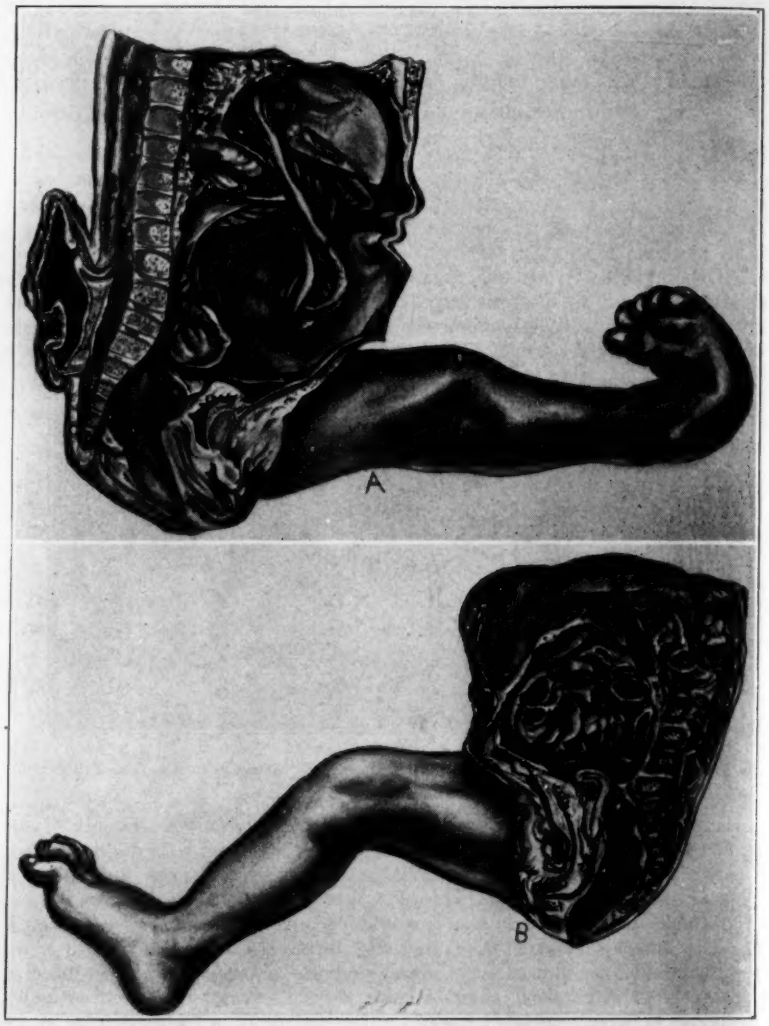

Fig. 2.—*A*, left half of the lower part of the trunk of an infant with prolapse of the uterus, meningocele and clubfoot. *B*, section of the lower half of the trunk of a normally developed premature stillborn female infant for comparison with the pathologic condition shown in *A*.

It has been shown [2] that occult spina bifida may be associated with prolapse of the uterus in women and may be a factor in its causation, especially in the nullipara.

University of Georgia School of Medicine.

2. (*a*) Von Graff, E.: Etiology of Prolapse, Am. J. Obst. & Gynec. **25**:800, 1933. (*b*) Laws, G. M.: Consideration of Lumbosacral Spina Bifida Occulta with Special Reference to Uterine Prolapse, ibid. **33**:126, 1937.

DOUBLE UTERUS, VAGINA AND RECTUM

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We recently saw a 12 year old girl whose complaint was fecal incontinence since birth. Her combination of anomalies was unique in our experience, and a search in the literature has failed to reveal any report of a duplicate condition. The rarity of the abnormality and the success of the treatment devised to remedy the distressing incontinence make it worthy of report.

REPORT OF A CASE

C. L. H., a 12 5/12 year old school girl, was referred to one of us (W. E. L.) in the summer of 1942 with the complaint of fecal incontinence from birth. The child was born at the Providence Lying-In Hospital, Providence, R. I., at full term by normal delivery. Her weight at birth was 7 pounds 6 ounces (3,348 Gm.). The infant was of good color, and there were no postnatal disturbances.

On the third day after birth she was first noted to have some abnormality of her vaginal introitus in that feces appeared at the vaginal outlet. Since the infant had normal bowel movements through a normal anal opening, no immediate treatment was recommended. The family physician diagnosed a rectovaginal fistula and advised deferring any operation until the child was larger.

On June 21, 1937, at 7 years of age, the patient was admitted to the Memorial Hospital, Pawtucket, R. I., with the diagnosis of a pelvic mass outside the rectum. The patient was examined under nitrous oxide–oxygen–ether anesthesia, and a mass was found which seemed to be in the vaginal septum between the rectum and what was thought to be the vagina. A roentgen examination showed that the large bowel was filled with irregular collections of gas, but changes were not sufficient to warrant a diagnosis of intestinal obstruction. The pelvis was filled with a mass suggestive of impacted feces, which presumably was anterior to the rectum. Roentgenograms taken the following day, after a barium sulfate enema given rectally, showed the rectum compressed anteriorly and displaced to the right side by a mass in front of it. No fistulous connection with the vagina or the space anterior to the rectum was demonstrated. The remainder of the colon and the terminal portion of the ileum were normal.

On June 25 hippuran solution was injected into the so-called vaginal sinus; the dye diffused through a large collection of feces, measuring 4 by 3 inches (10 by 7.5 cm.). This mass was pear shaped and occupied a position in the center and slightly to the left of the midline. At no time was a connection demonstrated between this mass and the rectum or the adjacent small bowel.

On June 28, with the patient under nitrous oxide–oxygen–ether anesthesia, the central opening in front of the anus was enlarged by sharp dissection to admit the examining finger. Large, hard fecal concretions filled this space. These were entirely scooped out. Only then could one detect a hard mass in the right lower quadrant and right vault, which suggested a solid ovarian tumor.

On July 1 an exploratory laparotomy was carried out through a midline incision from the umbilicus to the pelvis. It was reported that within the peritoneal cavity an exceedingly redundant sigmoid with a long, broad mesentery was found, which resulted in a partially twisted sigmoid. The mass in the right vault proved to be another fecal impaction, in the lower sigmoid. A small underdeveloped uterus was seen. The tube and ovary were identified on the left side, but neither was seen on the right. An examining finger

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in the rectum did not come in contact with the fecal mass, but a milking manipulation from above forced this mass downward and it presented into the so-called vagina. Since this effected some shock, the incision was closed without further exploration.

The recovery from the laparotomy was uneventful, and the patient was discharged on the sixteenth postoperative day, improved, but not cured of her primary condition.

Beginning in the summer of 1942 the patient suffered from dysuria, with frequent urination, six times daily and two or three times each night. Control of the urinary sphincter was poor. Urinalysis detected pus which cleared readily with sulfanilamide therapy. More bowel movements came through the abnormal anterior opening than through the anus. The family doctor recommended vaginal douches of mild protein silver, and the use of a perineal pad; he referred the patient to one of us (W. E. L.) for treatment of the incontinence.

On Aug. 10, 1942 the patient was admitted to the private ward of the Children's Hospital. At that time her temperature was 98.6 F., the pulse rate 96, the respiratory rate 70 and the blood pressure 122 systolic and 70 diastolic. She weighed 102 pounds (46.3 Kg.), appeared to be well developed and well nourished and showed early signs of maturation.

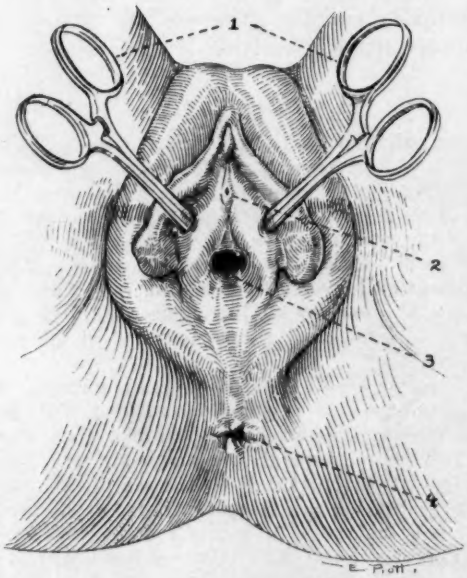

Fig. 1.—Drawing of external genitalia: *1,* Kelly clamps inserted into the lateral vaginal orifices; *2,* urethra; *3,* so-called vaginal outlet; *4,* anal orifice.

Her head, ears, eyes and nose were normal. The tonsils were enlarged but not inflamed. The neck was normal, the chest symmetric and the breasts firm, moderately rounded and not unusual. The lungs were normal, and the heart was not enlarged, but there was a soft, blowing systolic murmur best heard at the base. The abdomen was rounded, soft and free of masses, tenderness and spasm. There was a well healed, long suprapubic midline scar. The pubic hair was soft and abundant. The labia majora and labia minora were normal. When these were adequately spread apart the urethral outlet seemed surrounded by redundant folds of mucous membrane. When a no. 12 catheter was introduced centrally among these folds clear urine was obtained. Just lateral and posterior to the urethral opening were two other openings, the significance of which was not at first clear. The so-called vaginal outlet measured 1.5 cm. in diameter and was normally placed, in the midline, between the urethra and the anus. The anal orifice was normal in appearance, and the sphincter was of good tone.

Three days after the patient's admission, when the rectum had been rendered empty by rectal irrigations and a low residue diet, a more thorough examination could be made, which showed the condition depicted in figure 1.

Among the redundant folds of labial mucous membrane three orifices were identified. Anteriorly, at the base of the anterior fourchet, was a urethral outlet which seemed counter-

sunk. Into this a no. 14 French catheter was inserted with ease. Clear urine was obtained which when cultured was found to contain Bacillus proteus. Just posterior and lateral to this orifice were two accessory openings. These appeared 1.25 cm. in diameter and were surrounded by irregular folds of mucous membrane. A no. 20 French catheter could be inserted for 10.5 to 11 cm. before obstruction was encountered. No fluid was obtained with the catheter. The interpretation of these accessory orifices was deferred.

By rectal examination the perineal body was found to be of good consistency and size. Bimanual examination with one finger in the so-called vagina and the other in the rectum disclosed the presence of an extremely thin septum between the two lumens. No connection between these two canals could be felt. No. 14 rubber tubing was fed into the so-called vagina without obstruction, and it returned with its tip covered with feces. Fluid introduced through this catheter came out through the rectum. Somewhere beyond the reach of the finger tip there was a connection between these two lumens. Proctoscopic and sigmoidoscopic examinations were next carried out. The medium-sized sigmoidoscope was introduced through the anus for a distance of 24 cm. and then withdrawn slowly; no abnormality was observed. Subsequently, the small sigmoidoscope was introduced into the so-called vagina, where it passed upward 16 cm. with ease and showed only normal-appearing colonic mucous membrane. It was believed that the instrument had passed through the rectovaginal fistula into the sigmoid.

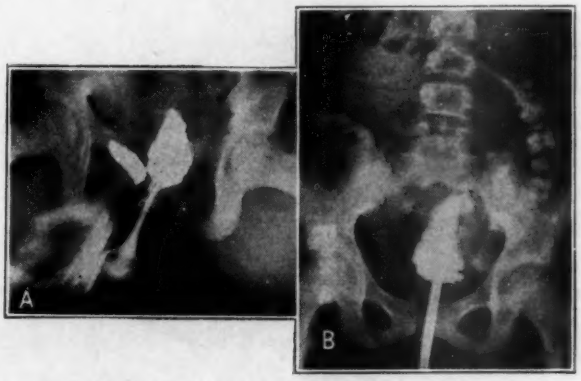

Fig. 2.—*A*, roentgenogram (after injection of iodized poppyseed oil) showing right and left laterally placed vaginas. *B*, roentgenogram taken after injection of barium sulfate into the centrally placed so-called vagina. Note how the column of barium quickly advanced from the so-called vagina into the sigmoid.

Bimanual examination with a finger in the so-called vagina and a hand on the abdomen was unsatisfactory, since the patient's abdominal musculature was not relaxed. The uterus, tubes and ovaries were not made out with any degree of certainty.

During the afternoon of the same day, several roentgenologic studies were carried out. A cystogram was normal. Iodized poppy-seed oil 40 per cent injected into the accessory lateral orifices demonstrated blind pear-shaped pouches which measured 12 by 5 cm. (fig. 2*A*). Fluoroscopic examination of the so-called vagina after introduction of barium sulfate proved a connection with the sigmoid (fig. 2*B*). Films of the pelvis and the lumbosacral portion of the spine revealed an early spondylolisthesis at the lumbosacral junction. Two days later intravenous pyelograms showed prompt appearance of the dye bilaterally. The left renal pelvis tended to be bifid. A semilunar depression of the inferior calyx was seen on the right side. There were no other abnormalities.

On August 14 the patient was sent home, to return later for exploratory laparotomy. We knew there was congenital abnormality, but whether this consisted of accessory vaginal pouches, a rectovaginal fistula or a duplication of the rectum was not clearly understood. A suprapubic exploration was essential before any reparative work could be advised.

On September 17 the patient was readmitted to the hospital. The following morning exploratory laparotomy was carried out with the patient under nitrous oxide–oxygen–ether anesthesia. The abnormalities observed in the abdomen are graphically recorded in figure 3. A long midline suprapubic incision was made, excising the old scar. The liver, spleen and kidney felt normal. The cecum and appendix were normally rotated and fixed in the right lower quadrant. The colon appeared normal. The sigmoid was unusually redundant and

had a persisting remnant of the anterior mesentery fixing its lower half to the posterior wall of the bladder. This mesenteric veil was completely reflected to the floor of the pelvis, in such a way as to divide the latter in half. Once the loops of bowel were walled off with sponges, the contents of the pelvis were identified. A normal-sized, pear-shaped uterus was present on each side. Each uterus had a single tube, ovary and round ligament. The ovaries were normal. On the left side there was a hydrosalpynx which measured about 5 by 5 by 14 cm. and which contained clear fluid. The terminal portion of the sigmoid, above the pelvic floor, was of normal caliber. When a long rubber catheter was inserted into the so-called vagina and another into the rectum, both these structures could be identified as entering a single lumen of intestine just below the pelvic peritoneal reflection, at a point approximately 16 cm. above their external outlets.

At this juncture we felt we had identified two uteri, two vaginas with laterally placed vaginal outlets, and a duplication of sigmoid and rectum with two external openings.

Fig. 3.—Drawing of internal genitalia: *1*, left hydrosalpynx; *2*, right ovary and tube; *3*, laterally placed uteri; *4*, laterally placed cervices; *5*, anterior mesentery forming a septum which divides the pelvis into halves; *6*, bladder.

The postoperative course after this exploration was uneventful. Irrigations of the rectum and of the sigmoidorectal duplication were carried out three times a day on the ninth and on the tenth postoperative day.

On the eleventh postoperative day the patient was submitted to a perineal plastic procedure to short-circuit the feces which descended from the upper fork of the duplicated sigmoid and discharged from the central so-called vaginal outlet. A circular incision was made in the perineum around the anterior portion of the duplication. This anterior branch of the duplication was then freed from its opening down to the point where it was in close approximation to the posterior portion of the rectum. An opening was now made in the posterior portion of the rectum, and the anterior portion of the duplication was anastomosed to this opening so that the fecal content of the anterior portion of the duplication would empty into the rectum. This procedure provided not only the rectum but the duplication

with adequate sphincter control of the normal anus. The defect in the perineum which was caused by the dissection was now closed with interrupted sutures, a good perineal body being left. The patient was put on a low residue diet, and peristalsis was limited by the use of camphorated tincture of opium and codeine. Administration of liquid petrolatum by mouth was started on the fifth postoperative day, and on the sixth day the patient had her first soft bowel movements by rectum. On the eighteenth postoperative day there was a small leak of liquid petrolatum through the perineal incision. On examination by rectum it was felt that the new stoma was not of sufficient size.

On the twenty-second day after this first plastic procedure a second operation was carried out, to enlarge the stoma. A midline incision was made in the thin septum between the rectum and the duplication, and the mucous membranes of the duplication and the rectum were sewed together to make a larger opening, 7 or 8 cm. in length. The result

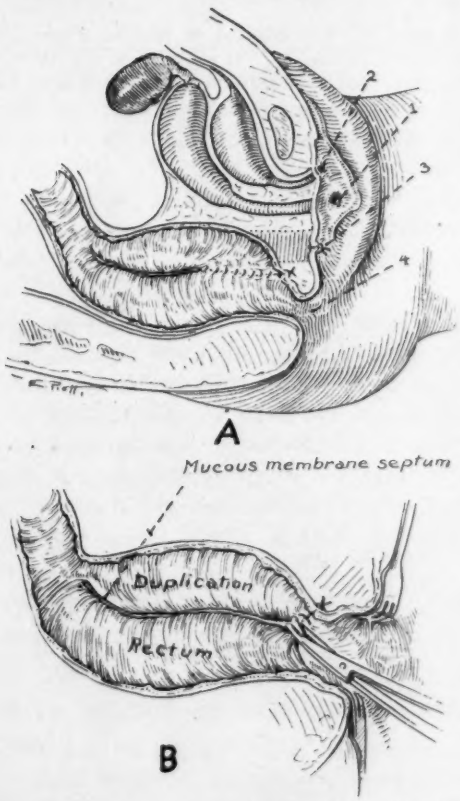

Fig. 4.—*A*, sagittal section showing the final operative result: *1*, laterally placed vaginal outlet; *2*, urethral meatus; *3*, site of closure of the duplication; *4*, anal orifice with an artificially arranged stoma connecting the rectum and its duplication just inside. *B*, insert showing how the thin rectal septum was split to enlarge the stoma.

of this procedure was extremely satisfactory (figs. 4 *A* and 4 *B*). The perineal wound healed completely and the patient had complete control of defecation, having one or two normal bowel movements each day. When the patient was seen one month after discharge from the hospital, the result had remained entirely satisfactory, with perfect fecal continence and no tendency for fecal impaction to develop in the duplication.

COMMENT

This combination of anomalies presumably represents an arrest in the development of the uterus, vagina and rectum. The duplication of the rectum probably represents a schism of this portion of the bowel taking place during the transition

from the solid stage to the recanalized form.[1] The external opening of the duplication possibly represents a remnant of the cloaca. The two lateral vaginas are the result of an inhibition of the fusion of the müllerian ducts, which is also responsible for the duplex uterus.[2]

The operation devised for the relief of the incontinence has proved effective and is certainly safer and more feasible than resection of the duplication could possibly be.

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1. Keith, A.: Human Embryology and Morphology, ed. 5, Baltimore, William Wood & Company, chap. 21 and 23.

2. (*a*) Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2, chap. 19. (*b*) McBride, R. W.: Double Uterus, Double Cervix and Double Vagina, Am. J. Obst. & Gynec. **20**:710 (Nov.) 1930.

Progress in Pediatrics

POLIOMYELITIS

I. BASIC CONSIDERATIONS NEEDED FOR JUDGING THERAPEUTIC RESULTS

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This communication concerns itself chiefly with clinical and pathologic observations in poliomyelitis and the probable deductions which may be made therefrom. In later articles I shall describe the methods of treatment and report the results for patients cared for in the Division of Contagious Diseases, City Hospital, Cleveland, during the epidemics of 1941 and 1942. These will be compared with the results of McCarroll, Cole and Knapp, Stimson and associates, the Kendalls, Stone and others. In a final review, I shall try to analyze the recent literature on poliomyelitis.

PORTAL OF ENTRY

The available evidence suggests that the virus has its portal of entry anywhere along the gastrointestinal tract from the upper portion of the stomodeum down through the lower reaches of the intestine, usually from the midgut. During a mild epidemic, the patients with paresis or paralysis usually have involvement of the legs or arms, probably a spread from the midgut and along the sympathetic chain to the lumbar portion of the spinal cord; in a certain proportion of the patients the spread is along the tenth nerve (probably vagus neuritis at first) or along other cranial nerves to the bulb. Rarely, if ever, does the virus enter by way of the olfactory mucosa.

In severe epidemics there will be more persons with severe involvements. In recent years there has been an increase in the number of patients with paralysis of the ninth nerve.

ROUTE TO THE CENTRAL NERVOUS SYSTEM

The virus is picked up by the gray-fibered peripheral nerve endings of the autonomic nervous system, whether afferent or efferent, and routed directly to the spinal cord or midbrain by way of the axis-cylinders. There is no reason why virus cannot travel dromically or antidromically. The virus may be compared to a river overflowing its banks. If there is a minor flood, the virus covers merely the immediate lowlands (lumbar area); if a greater one, it backs up and inundates higher territory (cervical area) as well. Again, the flood may be so great as to affect distant areas (bulbar area). It may also course over the seventh, ninth and tenth nerves to the bulb.

OBSTACLES TO A "TAKE," OR SUCCESSFUL PRODUCTION OF THE DISEASE

Most human beings have some protection against the virus, so that even though they may harbor the agent of infantile paralysis they will be spared. Thus, even

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though the virus is present in the gastrointestinal tract and has approximated the axis-cylinders one may still escape the disease. However, there must be an unbroken connection between the absorbing axis-cylinders and the central nervous system, and the distance between these two points must not be too great, since the virus may be absorbed and excreted long before it can reach the cord and do objective damage. The spread of the virus may be stopped at the periphery of the nerve or somewhere along the pathway of the nerve fibers, or it may die out before reaching the cord.

Rapidity of absorption, ease of spread, obstacles to transmission and virulence of the strain are all factors to be considered.

PATHOLOGY

The absorbing virus does not cause any demonstrable reaction in the axis-cylinders that can be appreciated by present day standard methods of examination. (It is felt that there may be some evidence when polariscopic examination is made.)

When virus is injected subserosally into Macaca mulatta monkeys, it spreads along the sympathetic nervous system in the manner referred to previously. Polariscopic examination may show involvement of the peripheral somatic nerves. Twenty-four hours after the injection there are lesions in the cells of the autonomic and spinal ganglions, and only a little later there are lesions in the cord, involving not only the anterior horn cells but the cells of the stroma and parenchyma in the lateral and posterior portions.

Any area of the central nervous system, sensory, motor or autonomic, may be involved, although the cells about which there is most concern are those of the anterior horn area, i. e., the cells of the lower motor neurons, whether in the cord or in the medulla.

Karyolysis, neuronophagia, cellular infiltration with lymphocytes or leukocytes, crowding of Virchow's spaces and perivascular cuffing with lymphocytes may be noted. This is the classic type of reaction.

It is believed, however, that the anterior horn cells are first destroyed and Nissl's substance lost; then follow fading, shrinking and karyolysis of the cells. These are associated with some edema and congestion. Neuronophagia accompanies or closely follows this reaction, and there may be an early increase in polymorphonuclear cells. These changes constitute the primary phenomenon.

Other typical pathologic reactions, such as increased vascularity, perivascular cuffing, lymphocytic response and localized collections of red cells, are secondary phenomena and need not necessarily be present to permit a definite diagnosis.

There may be meningeal reactions.

Thus, the pathologic changes are such that the autonomic, sensory and motor systems are involved as well as the meninges.

Some authors, on the other hand, have felt that the vascular phenomena occur first and that cellular destruction follows. The first opinion is more logical and is now more widely held. It is admitted, however, that vascular phenomena in themselves may cause cellular destruction by pressure. Whatever the belief, the pathologic end result may be the same.

Condition of the Anterior Horn Cells.—1. In any given area of the cord neurons may be destroyed and yet there may be no objective clinical evidence in the periphery that disease is present. The neurons remaining have the power and sufficient connections to activate the muscles.

2. There may be destruction of many neurons and some peripheral evidence of disease. The fibers innervated by the cell neurons destroyed will not regenerate,

but the muscle fibers that are still innervated may hypertrophy. When this happens, their bulk may with exercise become like that of the comparable normal muscle and, in addition, movement of the muscle may actually become normal.

3. A few neurons and their axis-cylinders may remain although the muscles supplied by a particular segment may appear to be completely paralyzed. The muscles cannot be moved voluntarily. The impulse traveling from the cord to the muscle along the few surviving fibers is insufficient to move the muscle; the functioning muscle fibers do not react to their best capacity because braked by some condition, probably in the muscle; the muscle itself lies completely flaccid. Patients with such an involvement may show improvement with muscle training.

The foregoing conclusions are the result of observations and study of: (1) the experimental disease in monkeys as compared with the pathologic observations at autopsy; (2) pathologic specimens obtained from a patient who recovered from poliomyelitis and who died a few weeks thereafter from another disease; (3) clinical experiments with infra-red heat on patients with paralysis of an isolated muscle (involvement of the seventh nerve).

There must be in the spinal cord, as in the kidneys, liver and other organs, a vast oversupply of functioning tissue. The extent of the pathologic changes in the cord bears no absolute relationship to the clinical paralyses. A few neurons with their axis-cylinders may take over the function of the entire nerve. However, these few fibers in an apparently completely paralyzed muscle can take over the function of other fibers only if the muscle is kept in good condition, so that it does not offer any resistance to projection of impulses. This is best accomplished by keeping the blood vascular supply more than adequate. This fact has been demonstrated.

No such artificial distinction of types as "paralytic" and "nonparalytic" can be made except clinically. From a pathologic standpoint, it is safe to assume that in any patient with clinical symptoms of poliomyelitis there will be some pathologic reaction in the cord and that this reaction will usually be more severe than peripheral symptoms seem to indicate.

Secondary Pathologic Changes.—The paralysis of the muscle found in poliomyelitis may be due either to an involvement of the anterior horn cells or to the edema which temporarily causes diminution or loss of function in these cells. The capillaries are said to be increased in the gray-fibered matrix of the spinal cord, although it is my impression that in this condition previously existing vessels become dilated and hyperemic and, consequently, more apparent, rather than increased in number. In any event, when there is capillary dilatation there may be diapedesis of formed or fluid elements into the tissues. Single, multiple or extensive single hemorrhages may be found in gray-fibered areas of the spinal cord, particularly in the area of the anterior horn cells and in the pars intermedia.

If the capillaries of the cord are dilated, even simple oozing or further diapedesis of otherwise normal physiologic material into the crowded cord tissue may result in paralysis, solely because of pressure. If it is continued for some length of time, such edema and pressure will cause destruction of the nerve cells. It is important to remember that this disease is located in an organ where a maximum of clinical damage can be produced by a minimal pathologic lesion, a fact which must be borne in mind when treatment is considered.

Remote Effects.—Wallerian degeneration of the nerves peripheral to the lesion, atrophy of the muscles and hyperplasia of Peyer's patches are found. Other conditions noted are insignificant.

POINTS OF EPIDEMIOLOGIC INTEREST

Poliomyelitis virus probably can be found in the stools of nonafflicted and nonexposed persons. Neutralizing antibodies have been demonstrated in the blood serums of persons who never heard of the disease and of persons living in quarters where the disease had never occurred. Virus may be found in the stools of many nonafflicted but exposed persons. The evidence points one way, although not all the links in its chain have been forged. It may be said that during an epidemic many persons harbor the virus in their gastrointestinal tracts but that only a scant few of them acquire the disease. In brief, poliomyelitis is naught else than an accident occurring during the process of mass exposure.

The finding of the virus on flies is of little practical importance; if it should be found in rats (or other small animals) or on food, this fact would go far to explain the confusing epidemiology.

CLASSIFICATION OF PATIENTS

Persons in whom the virus of poliomyelitis is present may be conveniently divided into groups on the basis of the presence or absence of symptoms and signs of the disease:

Group 1. Those who have the virus in the gastrointestinal tract and who exhibit no symptoms. The number in this group will probably be great during epidemics.

Group 2. Those with virus in the gastrointestinal tract who merely have gastroenteritis (diarrhea or constipation), headache or some other nonspecific disturbance and no other symptoms.

Group 3. Those who have the aforementioned symptoms plus nausea and even pain in the belly. Groups 2 and 3 have what are termed abortive forms of the disease, and these patients too may be considered numerous.

Group 4. Those who have symptoms and significant changes in the spinal fluid but no paresis or paralysis.

Group 5. Those who in addition to the aforementioned symptoms have a positive Kernig sign (tightness of the muscles; hamstring tension of the posterior muscles of the hamstring group), a stiff back and no paresis or paralysis.

Group 6. Those who, in addition to a positive Kernig and a positive Brudzinski sign and a stiff back, have a stiff neck, some with marked opisthotonos. Groups 4, 5 and 6 are sometimes classified as "nonparalytic." They have no paresis or paralysis.

Group 7. Those who have all the previous signs and symptoms plus some segmental muscular paresis, which can be appreciated only on careful examination—a paresis not accompanied by any functional impairment of synchronous movement.

Patients in groups 2 to 7 inclusive arrive in a hospital for persons with contagious disease and usually recover within the isolation period of twenty-one days and are discharged cured. They rarely need to see an orthopedic surgeon; they rarely require any physical therapy other than that received in the hospital.

Group 8. Those who have all the signs and symptoms previously mentioned plus some obvious segmental weakness which does not fully approach paralysis and which does not interfere with synchronous rhythmic motion.

Group 9. Those who in addition to all the aforementioned signs and symptoms have a typical lower motor neuron lesion with segmental paralysis. Years ago it was rare that a patient got into a hospital unless he belonged to this group. At

the present time, in communities which have become conscious of poliomyelitis conditions of diagnosis have so improved that in epidemics many patients with abortive, nonparalytic forms of the disease and patients with paresis belonging in groups 2 to 7 inclusive are recognized. They may constitute the majority of persons that are admitted to the hospital during an epidemic; in one of the epidemics in Cleveland they made up nearly 80 per cent of the patients, and in no epidemic have they constituted less than 30 per cent.

The patients in groups 8 and 9, those with paresis or paralysis, are admitted to the hospital and later cared for by the orthopedic surgeon and the physical therapeutist. In the future these will still be the only patients whom these specialists will see or in whom they will be interested.

Group 9 must be subdivided, since the segmental paralysis present may occur in areas which give distinctive and characteristically different clinical reactions.

(*a*) Patients with the usual segmental type of disease with involvement of the somatic nerves and paralysis of the peripheral muscles.

(*b*) Patients with involvement of the phrenic and intercostal nerves, with respiratory distress, solely or in combination with spinal paralyses.

(*c*) Patients with lower motor neuron lesions of the cranial nerves, usually of the ninth and tenth nerves, with bulbar paralysis, solely or in combination with spinal paralyses.

(*d*) Patients with palsy of the seventh nerve, solely or in combination with spinal paralyses.

(*e*) Patients with combinations of encephalitic and bulbar signs and perhaps peripheral segmental disease as well, the polioencephalitic type (rare). In addition, there may be patients with uncomplicated peripheral neuritic, hemiplegic or cortical involvement as the sole evidence of disease. This is not common. Landry's disease is mentioned here merely to complete the list.

In a report on results, abortive, nonparalytic, bulbar, encephalitic (rare), hemiplegic and peripheral types of poliomyelitis should be especially noted; results attained in cases of these types should be kept separate from the results obtained in cases of poliomyelitis with objective paresis or paralysis of the muscles of the trunk, arms and legs.

One should first decide whether as a result of treatment any one member of group 9 *a* could be taken out of this group and be placed in either group 7 or 8. Such a test is a severe one indeed, but it quickly teaches one a sense of proportion, some modesty and, above all, deep humility, for many of the patients in group 9 *a* will still be in 9 *a*. This, however, does not give information as to the general condition of the patient as a whole. It does not mean that he is not better, and therefore it does not truly gage the functional results obtained with therapy.

It is most desirable to have patients relaxed, and the degree of passive mobility should be noted at the beginning, i. e., two weeks after the onset of the disease, and later, when the results of therapy are being evaluated. Passive mobility may be of little use to the patient if the muscles are still completely paralyzed. Then it becomes a question as to whether the muscles should be allowed to remain passively relaxed or whether it might not be better in some instances to get the arm, ankle, etc., fixed and ankylosed in positions of greater mechanical advantage.

As stated previously, the patient should be examined on his admission to the hospital, in order to get an estimation of his condition. When results of therapy are to be gaged, the patient must be examined for his base line condition two weeks after coming into the hospital. This status is compared with the status

observed at subsequent intervals when the therapeutic results are to be evaluated. For comparisons, patients may be classified somewhat as follows:

Status 1. Patients in whom no weakness or paralysis in any group of muscles is evident. (At the onset this would include patients with abortive paralysis and nonparalyzed patients.) Patients may be in status 1 on subsequent examination, and this group then would include many that originally were in a more advanced status but who had completely recovered. These patients would have passive movement through a normal arc of motion.

Status 2. Patients whose gait and balance are normal and who appear to have unimpaired use of all extremities. Careful examination, however, reveals slight weakness in one or more groups of muscles. (These patients are functionally normal but have to be watched for substitution. If the patient has moved from a more severe status to status 2 on reexamination, he is functionally cured.) These patients would have passive movements through a normal arc of motion.

Status 3. Patients with severe paralysis of one extremity and/or moderate paralysis of several extremities. These impairments are such that with the aid of braces, crutches or other supports the patient is able to get around relatively well. This may be the picture on the first or on some subsequent examination. These patients would have passive movement through a normal arc of motion.

Status 4. Patients with severe paralysis of the muscles in two or more extremities. These involvements are such that the patient is not (or will not be) bedridden but even with the use of the proper supports has (or will have) only limited activity. This may be the picture on the first or on some subsequent examination. These patients may be (*a*) passively mobile or (*b*) fixed to achieve some mechanical advantage.

Status 5. Patients with such severe paralysis of their extremities that they are bedridden. These patients may be (*a*) passively mobile or (*b*) fixed to achieve some mechanical advantage.

Unless some such comparison is made there can be no meeting of minds and no critical interpretation of results.

DIAGNOSIS

1. A diagnosis must never be considered absolutely proved in the absence of positive findings in the spinal fluid.

2. No persons with paresis or paralysis of the muscles should be considered as having poliomyelitis if the involvement is not segmental.

3. The lesion in this disease must have the characteristics of a lower motor neuron lesion.

4. Despite the presence of paralysis, no persons with distinctive sensory findings (dissociation of temperature, pain sense, etc.) or signs of an upper motor neuron lesion should be considered as having poliomyelitis.

Patients should be considered separately and according to types, for unless this is done marked beneficial results can be ascribed to some therapeutic procedure when the patients treated may not even have had poliomyelitis. They may have had a disease from which in due course they would have recovered anyway, such as peripheral neuropathy.

Mistakes in Diagnosis.—Many mistakes are made in diagnosing poliomyelitis. Fully 50 per cent of the patients seen by the physician during the hysteria of an epidemic have nothing wrong with them or something other than infantile paralysis. Even many patients seen by physicians and sent to the hospital do not have the disease.

(*a*) The most common condition seen in children or in adults is weakness associated with fever. This may accompany any contagious or infectious disease. Sometimes it is so marked that the parents and even the physician are misled and feel that the patient may have infantile paralysis. One can easily see how a good prognosis can be given with any therapy if diagnosis is not accurate.

(*b*) Rheumatic fever is often mistaken for infantile paralysis. The first thing a physician should determine is whether the particular muscle or muscles involved are not being moved (1) because the patient is unable to move them or (2) because he does not want to move them on account of pain. Some patients with rheumatic fever have actually been reported as having poliomyelitis, especially when lumbar punctures have not been done. They usually improve with rest in bed. Consequently, if they are considered as having poliomyelitis and treated by any method, the outcome seems to represent a remarkable therapeutic result. This demonstrates the importance of early examination.

(*c*) There is no disease which causes weakness so quickly as influenza, especially when it is associated with gastrointestinal symptoms. It is often mistaken for poliomyelitis, or vice versa. Since the intestine is often involved in influenza also, the early symptoms may be similar and the peripheral weakness will be suggestive. Such manifestations have been classified as poliomyelitis—a thing perfectly possible if no laboratory work has been done and a meticulous clinical examination has not been made.

(*d*) Any one of the encephalitides may involve paresis or paralysis, as can also rabies, measles, herpes zoster and other conditions. Some diseases which produce symptoms and cause lymphocytes to appear in the spinal fluid may be confused with poliomyelitis—diseases such as mumps, whooping cough, postvaccinal encephalitis, benign lymphocytic choriomeningitis, the St. Louis type of encephalitis, idiopathic encephalitis and disseminated encephalomyelitis.

(*e*) Myotonias should easily be ruled out on the basis of history and results of physical examination.

(*f*) Acute arthritides may also lead to an incorrect diagnosis.

(*g*) "Peripheral neuropathy" or neuritis has to be differentiated. With this condition there may be a simple involvement of one peripheral nerve or massive involvement of all branches of a large nerve, and slight and no objective involvement or massive sensory involvement seemingly disassociated, especially as regards pain and temperature sense. Both peripheral neuropathy and poliomyelitis are lesions of the lower motor neuron, poliomyelitis of the cells themselves and peripheral neuropathy of the nerve trunk. A peripheral neuropathy may begin in the extreme periphery of a nerve. It may stop spreading midway to the cord or may actually extend up and involve the whole nerve, so that at times it becomes difficult to exclude infantile paralysis.

One cannot always utilize sensation to discriminate between poliomyelitis and peripheral neuropathy, because in my experience striking objective sensory symptoms in peripheral neuritis are not the rule. These patients may have slight paresthesias, slight numbness and perhaps a slight sense of electric-like tingling. This is overshadowed by the severe pain which may be present in poliomyelitis in the early stages of the disease. The authors of textbooks who write about objective sensory symptoms probably have in mind one type of patients, but they certainly cannot speak for the majority of persons who have peripheral neuritis, in whom objective sensory changes are elicited with great trouble if at all. It is true, however, that these sensory changes may occur in those severely afflicted, especially in those with deficiencies, and the alcoholic neuritides (indirectly a deficiency). In severe peripheral neuritis there is found bilateral symmetric involvement in both

arms and legs; it may be bilateral in the arms only or in the legs only. The paresis or paralysis is initiated in the intrinsic peripheral muscles, and in the ordinary form the muscular paresis or paralysis becomes less severe as one goes toward the trunk. If there is a complete involvement of all the muscles of both legs or arms, one cannot in that one member differentiate this disease from poliomyelitis. The differentiation can be made only by studying the muscles of the other members of the body and looking for segmental involvement. If this is found, a diagnosis of infantile paralysis can be made. I have seen 2 patients with peripheral neuritis demonstrated as patients with infantile paralysis, patients who have made remarkable and amazing recoveries within a few months after having been completely paralyzed.

The patient with poliomyelitis in whom all nerve cells are involved will make no recovery despite any treatment. Nerve cells may be destroyed directly by virus or indirectly as the result of anemia due to edema, etc. If nerve tissue becomes anemic, it dies in a short time. Unless the anemia is relieved, it will leave the patient with permanent effects. Edema is absorbed within a few weeks, and the cord becomes quiescent. If a person has massive paralysis due to the virus of poliomyelitis and this paralysis persists after a month and flaccidity is complete, it is known from the very nature of things that there has been a massive destruction of anterior horn cells and recovery will be partial or will not result at all. If the paralysis persists more than a month, the nerve tissue will be completely destroyed and there will be no recovery except in a minor degree. Therefore, when one sees patients who have had complete paralysis of all extremities for a few months improve so that at the end of four or five months they are walking about without any muscular weakness, one can be certain of one thing—these patients did not have poliomyelitis; they had "peripheral neuropathy."

I have made the mistake of considering 1 such patient during a severe epidemic as having poliomyelitis. All the evidence was at hand, minor sensory changes, etc., but in accepting the observations of others, I made a glaring error of omission.

I have seen patients with the Guillain-Barré syndrome classified as having poliomyelitis, and even in the recent literature the question has been raised as to whether the disease may be similar to poliomyelitis or perhaps caused by the same virus. Although I believe that it is not, it is nevertheless uncertain that this syndrome is not caused by a virus; it is, however, fairly certain that it is not caused by the virus of poliomyelitis.

For patients with tumor a diagnosis of poliomyelitis may be made. I have twice made this mistake. The first error was one of commission. I examined the patient, who had some gastrointestinal symptoms and an increased number of lymphocytes in the spinal fluid, and came to the conclusion that his disease was a nonparalytic type of poliomyelitis. The diagnosis for the second patient was a mistake of omission, since I did not examine the child and accepted the details of examinations by other physicians. The latter patient had paralysis. Both diagnoses were confirmed.

CLINICAL CONSIDERATIONS

A stiff back is commonly associated with any condition in which there is direct or indirect irritation of the muscles of the back or of the nerves leading to them. Fractures of the back, sprains, Pott's disease, the many forms of meningitis and many encephalitides cause a slight or great amount of stiffness of the posterior muscles of the back. Another common finding is tightness of the hamstring muscles. Finally, stiffness of the neck may occur. All these conditions have long been recognized and described in the literature as a positive Kernig sign—stiff back, pain along the spine, stiff neck and opisthotonos. The exact cause of these reactions is problematic. It may be that there is an irritation of the thoracolumbar

outflow with reflex stiffening of the muscles supplied by somatic nerves. In a mild type of poliomyelitis, one gets the original positive Kernig reaction when the leg maneuver is used; if the disease is severe, a modified Kernig reaction can be obtained by lifting the head. These are the typical reactions with any form of meningeal irritation.

How long will the positive Kernig and Brudzinski reactions and the stiffness of the neck and back persist? These signs disappear within two days to three weeks after the patient is admitted to the hospital—usually within five to twenty-five days after the onset of symptoms. Tightness of the hamstring muscles may persist, not because of meningeal irritation but because of other conditions in the opposing muscle.

The patient has no pain when he rests in a position of comfort, a position he automatically and instinctively takes; he has pain only when the neck is bent forward or when the back or the legs are straightened. This is the meningeal component of the disease. The objective signs and symptoms are exactly the same as in any other form of meningitis, and, as with these disease conditions, no treatment is necessary for them.

Ill informed physicians may fall into the error of thinking that these symptoms cause disease and begin to treat symptoms. These signs may be absent in the severely paralyzed patients; they may be the only signs in the so-called nonparalytic patient.

Frequently I have seen an enthusiastic physician attempt to straighten the leg at right angles to the body and if the patient is unable to respond claim that this is evidence of disease, probably of infantile paralysis; if the patient has had the disease, he claims that this is evidence that he needs more treatment. If this is good reasoning, then every patient with meningitis must have a touch of poliomyelitis.

Not all persons have the same fluidity of movement; muscles may be too long or too short; skeletal growth may not have paralleled muscular growth. The person may be large and fat, short and dumpy or slim and thin; he may have a pot belly. There will be differences in the pelvic structures depending on the sex. Thus, it is natural to expect some limitations of motion in patients examined.

Often demonstrators point to tightness of the hamstring muscles, which may or may not be the result of infantile paralysis, and attempt to draw conclusions from this limitation of motion—often unconscious of the fact that neither they nor any one in the audience has better mobility.

No person is symmetrically built, as can be easily seen by comparing composite photographs of body structure, body build or even facies. No one has the same amount of comparable strength in both sides of the body. Strength is comparable within limits and depends on the activity of the individual and the particular muscles used. There is no person who has reached the age of 3 years that does not have some beginning limitations of his previous freedom of motion. A child can stick his toes into his mouth during infancy and young childhood, but as he grows older and tries to accomplish this movement he finds that the feat has become more difficult; as he reaches adulthood, it becomes nearly impossible. Practically all adults have some limitation of motion—some stiffness of the hamstring muscles, which will not allow the legs to be brought up beyond right angles to the body. One cannot attribute disease to a person who has what almost every one else has.

Could a slight change in the spinal column—lordosis or kyphosis—have been caused by an unrecognized attack of infantile paralysis when there has been no epidemic? Any one claiming this would reveal an abysmal ignorance of the

history and epidemiology of the disease. It amounts to a statement that crooked backs have been brought about by unrecognized subclinical attacks of infantile paralysis in areas where there have been no cases. This would mean that in certain localities where there are a fair number of persons with crooked backs massive epidemics have occurred without the presence of a single recognized case—a thing utterly improbable; in fact, impossible.

People may be pigeon toed, knock kneed or flat footed or may even have slight lordosis or scoliosis. They are asymmetric to begin with, and later habits do not tend to make them more straight. In fact, if anything, their habits tend to aggravate the previous condition.

To those who have accepted the concept of a "motor unit" it is hard to see how fibrillations could occur and be appreciated clinically, fibrillations that would be limited to muscle fibers and still be apparent to the observing eye. It is more probable that one is dealing with some form of vesiculation in which the irritated "motor unit" causes a twitching reaction which is easily appreciated. This is commonly observed in monkeys with experimental poliomyelitis, but it is not so commonly encountered in human patients, although it can occur. It usually does not last long.

Pain in Poliomyelitis.—There are various types of pain, and it is certain that they are not all produced by the same mechanism. The original pain that patients with poliomyelitis feel is that which precedes somatic paralysis. It is always segmental and usually appears in the nerve distribution of those muscles subsequently to be found paralyzed. Occasionally, however, the pain may be referred to another segment. In most instances this pain has disappeared before the patient arrives at the hospital.

Second, there is the pain associated with attempts to overcome the protective (?) flexion contraction present with any meningeal irritation. This pain has been alluded to; it does not trouble the patient unless there are attempts to elongate muscles by straightening the back, neck and hamstring muscles. If the patient is quiet and is not disturbed, he has no subjective pain. Meningeal reactions with stiffness of the posterior muscles do not cause subsequent paralysis. If patients with such conditions are included among patients treated, a high rate of recovery could be attributed to any particular therapy. In times past these patients probably were never admitted to the hospitals; they went around with their stiff necks and in most instances recovered completely. Now they are usually discharged from the contagious disease hospitals within one month after admission.

There is pain associated with attempts to stretch muscles that are in reflex tonic contractions. Gentle slow squeezing with pressures from light to heavy does not cause painful response in these muscles if care is exerted not to stretch the muscles. Quick squeezing of a muscle, however, may cause pain. Further flexion of the muscles, if this is possible, does not greatly increase the pain. The pain is apparent only on active or passive activation with stretching; it is not a type of pain that troubles the patient if he is left alone. It probably depends for its existence on stimulation of the stretch reflex. The reflex tonic contraction may come on synchronously with weakness or paralysis of the opposing muscle; it may develop later, after the paralysis is obvious, or it may never occur at all.

There is a type of pain brought about when attempts are made to stretch a muscle which is in a state of contraction due to replacement of muscle with fibrous tissue. This occurs relatively late, however.

There is also a type of pain which occurs when the patient is getting better, especially if his recovery proceeds too fast and he uses his muscles too vigorously.

This is more like the local pain seen in thromboangiitis obliterans and disappears with rest and massage.

Reflex Tonic Contraction.—Does everybody with infantile paralysis have reflex tonic contraction of the muscles opposing those showing paresis or paralysis? The answer is no. Any physician who has cared for a number of patients with infantile paralysis knows that some wake up in the morning paralyzed. In fact, the term "morning paralysis" has been given to this condition. Every physician has had patients that have "paresis or paralysis" and no reflex contraction.

Patients are often seen with paresis or paralysis before the opposite muscles begin to contract reflexly. Every one has seen patients who have paralysis and reflex tonic contraction whose muscles suddenly became flail within a week after the onset of the disease—a flailness which because of the short time could not have been due to the reflex contraction. Some physicians are astonished to find tightness of the hamstring muscles, despite the fact that this condition has been described for over a half-century.

When there is no need for a reflex response in the opposing muscle and the muscles have a dual (separate and independent) supply, there is no reflex tonic spasm, as may be seen when one studies the paralysis of muscles supplied by cranial nerves with crossed innervation.

In hundreds of monkeys given the disease, whether by brain, by nerve or by a portal of entry in the gastrointestinal tract, the first muscular evidence of the disease other than tremor is always paresis or paralysis.

Exteroceptive Sensations.—Under abnormal conditions, normal exteroceptive stimuli which ordinarily would cause only a normal response may be magnified by differences in the threshold value of the receptor organs so that they cause marked reactions. There is no disturbance in exteroceptive sensations in poliomyelitis as obtained by the present available means.

Normal exteroceptive sensations are easily excluded by covering the skin with paraffin. Pain is still found in the muscle when it is contracted. There is a subjective sensation of cold, and objectively the skin of the paralyzed areas may be cool; both manifestations are probably due to vasomotor phenomena secondary to involvement of the autonomic nervous system. These sensory findings are quite different from the appreciation of external cold and heat which all patients possess unimpaired.

Proprioceptive Sensations.—Much has been said about proprioceptive sensations. One might even begin to think that these sensations were lost. The patient's discriminative system appreciates pressure, judges accurately the shape of solid objects and gives him a correct knowledge of his position in space, a concept of posture. He never loses his proprioceptive sensation.

Treatments do not furnish better proprioceptive appreciation. The procedures are intended merely to employ what a patient already possesses and what he has never lost. The object is to try to stimulate proprioceptive sensation while trying to project a nerve impulse over the remaining afferent fibers. What the patient lacks is the ability to project a current of motion over the axis-cylinders of the few remaining uninvolved anterior horn cells, and there is no proprioceptive loss.

Sensation.—Sensory epicritic and protopathic sensations are all present. This seems incongruous in view of the damage to the sensory system. I am not sure that there are no changes. Certainly, some of the recent experiments of my colleagues and myself have suggested that although gross sensation is apparently normal there may be finer sensibilities which have not been heretofore shown by the means at our disposal.

Should this be borne out, there could be sensory disturbances not too noticeable because of the oversupply of sensory neurons.

Temperature.—The temperature is not usually high, averaging about 38 to 38.5 C. (100.4 to 101.3 F.). If the bowels and bladder are not involved, it is down to normal within a few days after the onset. In case these organs are involved, the temperature will be elevated until their functions return to normal. In many instances it never rises above normal.

Balance.—An estimation of his ability to balance himself is most important for the patient. Balance is more important than strength. One can walk with little muscular strength provided the muscles are in balance. A little additional strength on one side as compared with the other will unbalance the patient. A careful estimation of strength of the muscles of the head and thorax must be obtained. This information is especially important because it will be difficult to teach a patient how to walk if he cannot balance his head and thorax.

If he is a child and his head lolls backward or to one side, its weight may be such as to topple him over, even though he may have good thoracic muscles. Imbalance of thoracic muscles is also important, and it is necessary to know their condition.

Frequently a patient is properly balanced. He goes about his business and appears to the unobserving eye to be normal, yet in a short while, because he uses the muscles too much, he will be found to be weaker and will begin substituting muscles. Even if a patient is normal in appearance and has good synchronous movement, meticulous care should be taken to ascertain if his muscles are or are not weakened. A normal-appearing person with hidden weakness may begin to show substitution and wry movements under a regimen of restricted activity.

Condition of the Skin.—In some patients the skin of the paralyzed side may be colder to the touch of the examiner than that of the nonparalyzed side. This is an objective, not a subjective, observation. I have never seen a trophic ulcer develop in this disease per se. Patients may occasionally have pressure necrosis followed by local fibrosis and scarring where tight casts are applied, but this would occur with normal skin. Spontaneous trophic ulceration of the skin should immediately make one suspect that the proper diagnosis was not made. Although there may not be a trophic ulceration of the skin, there may be a trophic condition of the bones and of the entire leg and arm and a complete failure of the member to develop.

Involvement of the Bladder and Intestines.—There may be evidence of intestinal involvement in that the bowels are constipated and enemas are returned clear; there may be pain in the intestines due to gas pockets, relieved by enema; gurgling may not be heard over the abdomen, especially if the intestine is severely involved, and peristaltic waves may not be seen.

The bladder may be the seat of a retention with overflow dribbling due to paralysis of the detrusor muscle and of the sphincters of the neck. The bladder will fill, and its buckling on itself at the neck will cause the retention. It never quite empties itself, but, since the patient wets the bed, the parents and even the physician are sometimes satisfied that it is properly functioning. When filled to capacity, it is obvious to the observer as a tumor and is painful when palpated. The retention usually precedes but may occur with or following somatic paralysis. Sometimes this may be the sole abnormal condition observed. In such circumstances there is no obstruction to the passing of a catheter, and the urine is clear. The episode occurs during the summer or fall and is associated with other symptoms. Given these facts, the diagnosis is poliomyelitis until proved otherwise.

Time of Recovery of Autonomic Function.—No matter how severe the somatic paralysis, function of the bladder usually returns in from seven to fourteen days, provided the bladder is emptied three to four times daily. If retention is not relieved, there may be a spread of the disease. Function also returns to the paralyzed bowel within seven to twenty-one days, and the patient must have frequent evacuation of its contents.

The autonomic functions of the bladder and the intestines return no matter what forms of therapy are employed. In rare instances there may be paralysis of the sphincter muscles of the rectum. The prognosis is bad in such cases as far as these muscles are concerned, because this paralysis usually is associated with massive paralysis of other muscles supplied by the sacral distribution. If a patient has had a severe somatic paralysis together with paralysis of the bladder and after he recovers has difficulty in starting the flow of urine or slight difficulty in stopping the flow, it is nearly definite that he did not have infantile paralysis.

There may also be disturbance in secretion of the sweat glands; often the patient sweats more profusely over the affected area. Sweating over the affected area may be induced by pilocarpine, but epinephrine, a specific thoracolumbar stimulant, cannot stop it; this indicates a specific disturbance of the thoracolumbar outflow.

Condition of the Abdominal Muscles.—Some commotion has been raised and the statement has been made that some form of therapy is miraculous because no abdominal muscles are found paralyzed after its use. As a matter of fact, the muscles of the trunk are infrequently involved except in persons in whom paresis and paralysis can easily be demonstrated. The only common thing about the abdominal muscles is loss of the superficial abdominal reflexes, to which I first called attention years ago, although the deep metameric reflex is present in practically all instances. Patients whose abdominal muscles are involved are those in group 9a, and even in these involvement is infrequent. A patient with a stiff back does not wish to contract these muscles because there is pain when the muscles of the back are stretched. This does not constitute paralytic involvement. When the Kernig sign has disappeared, the patient contracts the muscles again.

Selection of Patients for Treatment.—Patients with destruction of the anterior horn cells, mild or severe as the involvement may be, are the ones who should be selected for treatment, especially if superiority is claimed for any particular mode of procedure. In addition, whether poliomyelitis is mild or severe the diagnosis should be made absolute and should never be considered proved in the absence of positive changes in the spinal fluid. There are so many causes of paralytic conditions that the diagnosis of poliomyelitis should never be made if the weakness or paralysis is not segmental. No beneficial results can be claimed for any therapy when it is applied to patients of groups 2 to and including 7.

There is no need to treat patients if they will get better without any treatment. One should not scare a patient who needs no treatment into getting treatment on the basis that paralysis might develop. This is a typical sales method for nostrums. Such patients rarely become paralyzed. Should paralysis be found later, there is no evidence that it was not previously present; perhaps there was a careless examination.

How Beneficial Effects Are Noted.—In order to ascertain whether any procedure has been of benefit to the patient, his condition at the time the disease was at its maximum must be known. If the extent of the initial paralysis is not known, then no one is entitled to draw any conclusions about his method of therapy. Therefore, an estimate of muscular damage should be made for every patient. Only when this is done may comparisons be made. An objection to this might be made on the grounds that the patient may have pain if examined, which might retard,

impede or harass recovery. This is nonsense. Some persons may state that they can feel where the paralysis is and that this is sufficient. It is not sufficient; it implies powers of divination possessed by none. It also allows credit for impossible results. Impressions can never replace thorough examination.

Muscle Testing.—Muscular strength should be tested at the onset and two weeks after the onset of the illness in order to gain some impression of the severity of involvement. At City Hospital, Cleveland, this is done by noting the response to galvanic current and the proportionate strength of muscles. Since any miraculous progress occurring during the first few weeks of the illness is due to absorption of material in the spinal cord and not to any treatment, we wait two weeks before making an absolute estimation of muscular involvement in terms of percentage. To be impersonal one should estimate the damage objectively in terms of response to galvanic current.

Reflexes.—The first thing that disappears in poliomyelitis is the superficial abdominal reflex, which I first stressed years ago. The disappearance of this reflex is practically always associated with involvement of the gastrointestinal tract. There may be modification of the metameric reflexes anywhere from hyperactivity to loss of activity. Hyperactive reflexes occur when the disease proceeds more slowly. If the preparalytic period is long enough, hyperactivity rather than hypoactivity is first found, but it is a hyperactivity which disappears with continuous stimulation. Flaccidity and loss of function may be present. Atrophy may come on later.

Stages of the Disease.—It is not always easy to know when the acute stage of the primary disease is over. It may be within two or three to approximately seven days. There is usually no extension of paralysis after two to seven days; on the average, the progression ceases nearer the second than the seventh day. For practical reasons and from the standpoint of therapy, the acute stage should be considered to be as long as any therapeutic procedure may benefit the muscles. The contagious period is arbitrarily placed at twenty-one days, although virus has been obtained from stools as long as one hundred and twenty-three days after the onset.

Orthopedic surgeons have been criticized because of the way they have treated patients in the acute stage of this disease. In fairness to them, it should be pointed out that they do not often see the patient before the quarantine period and acute phases are over.

Résumé of Clinical Reactions.—It is obvious that when poliomyelitis occurs it is associated with symptoms which might arise because of involvement of the centers of sensation, voluntary movement and autonomic functions as well as of the meninges. Briefly, in the severe type of the disease, there are signs of meningitis with stiffness of the posterior muscles (muscles of neck and back and hamstring muscles) that are variable in character and accompanied by evidence of a segmental involvement of lower motor neurons with temporary reflex contraction of opposing muscles accompanied by pain, disturbance of the sweat glands, involvement of the gastrointestinal tract, loss of motility and other symptoms.

PROGNOSIS

There are two things to consider: Is the patient going to die? Will the paralyzed muscles regain normal function? One should now note the paragraphs referring to the various groups of patients with the disease. No one in groups 1 to 8 dies. All of the deaths occur in those patients classified under group 9. No one dies who is in group 9*a*, with a segmental type of disease

(Landry's disease excepted). In group 9 *b* there may be many deaths, the patients drowning in their own secretions or suffocating. Respiratory paralysis may be associated with bulbar paralysis, and there may be other combinations of paralyses, which makes the prognosis even more grave.

The bulbar type, present in group 9 *c*, accounts for deaths; in some epidemics as many as 75 per cent of patients in this group die. Recently, however, this rate has dropped to about 30 per cent.

Most of the deaths occur in patients in groups 9 *b* and 9 *c*. Mucus collects in the throat in these patients and drifts to the back of the pharynx. They cannot cough it out; it gets into the trachea and bronchi; the patients become blue; they cannot breathe, begin to choke and may even suffocate. They make themselves worse by taking shallow breaths or by trying to hold their breath. When they are in a state of air hunger and can no longer refrain, they take a deep breath, insufflate mucus into the trachea and have an attack of extremely difficult breathing, coughing and cyanosis. The immediate results obtained in such patients are truly remarkable, almost unbelievable. All that needs to be done is to aspirate the mucus and place the patient in a dependent position for good drainage, to assure him that he is out of danger and to urge him to adopt a regular breathing rhythm. These patients, apparently in the last throes of respiratory involvement and looking as though they would die any moment, become calm and quiet in a few minutes.

Usually, if the patient with bulbar poliomyelitis weathers the storm for forty-eight hours he will recover unless complications develop. If the patient has involvement of an isolated cranial nerve, the chances for his recovery are good.

Patients in group 9 *d*, with sole paralysis of the facial nerve, do not die.

Patients in group 9 *e*, who have a combination of encephalitis and bulbar and peripheral segmental paralysis, often die.

I have seen only 1 patient with uncomplicated peripheral neuritis which I was certain was due to poliomyelitis virus, 6 patients with uncomplicated cortical poliomyelitis and 2 patients with uncomplicated hemiplegia (group 9 *f*). Even in the cases of the last 2 patients the episode was so brief as to make me question the diagnosis. In any event, the prognosis for all these patients is good.

The next consideration is the prognosis of muscular recovery. Here opinion is rife and argument loud. The patients in the groups from 1 to 7 inclusive can be forgotten. It is possible that patients with missed lesions may erroneously be classified in group 7, a mistake appreciated only later, on subsequent examination. This does not mean that there is a new grouping; it means merely that a condition previously present has been missed. Even then, the prognosis for muscular recovery is good.

The prognosis as regards groups 8 and 9 depends somewhat on the type of therapy employed and the possibilities of the therapy.

Factors Which Influence Prognosis in the Groups with Muscular Paralysis.—As far as muscles are concerned, there are many factors to influence the prognosis of a patient with infantile paralysis. If the patient has little or no mentality, he will have little or no recovery. If he is spoiled, it will be difficult to get the kind of cooperation that is necessary to produce good recovery. If he is lazy, feels sorry for himself, is pampered and gives up easily, there will be difficulties in concentration and determination. If he is too young to comprehend, most of the treatment will have to be passive. If he is near adult age and insists on leaving the hospital before it is time for him to become an ambulatory patient, he is bound to develop patterns of substitution. If he is an adult and must get back into the economy of things, if he cannot afford treatment or if there is no place for him to get treatment for the proper length of time, the same observation applies.

A person who has weak muscles and who persists in weight bearing is bound to substitute. One who has the time and has a place where he can stay flat until the muscles are reeducated to the point where they are efficient against gravity has a much better prognosis.

A person who spends three weeks in the contagious disease hospital and then is promptly sent home without much direction as to after-care will do badly no matter what kind of treatment was employed. One who after three weeks of quarantine is treated as an ambulatory patient does not do so well. The best prognosis is given when the physician has absolute control over the patient as long as necessary. This may be for a year or even two, at least to the point where he may train and educate the remaining muscles to their best possible advantage. A competent physical therapeutist should oversee the training.

RECOVERY

Complete Recovery.—It is obvious from what has been said that complete recovery can be claimed if the patient can put his muscles to the same average use as he did prior to having an attack of the disease.

Advanced Supertraining.—Some persons go beyond these criteria for complete recovery and by persistent muscle training produce a litheness little short of acrobatic, an unnecessary suppleness for a plantegrade animal, a suppleness which will be lost gradually as soon as the patient returns to his proper stride, unless he persist in this unnecessary training.

There is no doubt that patients treated this way appear startlingly mobile to the average unthinking physician who examines them; but if the physician stops to reflect he will soon remember that such conditions are not approximated in the average person, including himself and the demonstrator. Such training represents only persistence in the training of good muscles. It should be reiterated that complete recovery can be claimed if a patient is able to put his muscles to the same average use as he did prior to having an attack of the disease.

IDEAL TYPE OF THERAPY

Patients should be institutionalized and observed daily. The cost is not too important if the results are beneficial, but good results of a new therapy must be shown in the patients in groups 8 and 9 *a*. If other methods give equally good results and are less expensive, they become the methods of choice. Therapy must be considered with regard to its cost. Results should be proportionate to the effort.

Should not every patient be given treatment, regardless of to what group he belongs, on the basis that he might have paralysis or weakness later? Why? Since all but a minimum of these patients get better anyway and these few are those that have been misclassified and for whom a proper diagnosis could have been made had a meticulous physical examination been done, there is no necessity for unusual therapy. One must exercise some common sense and decide for oneself what treatment is necessary. The types of patients who need treatment are those who fall into groups 8 and 9, and anything that benefits or relieves them is of importance.

Statistics.—Conditions should be the same in order to be comparable. If any new therapy is presented, the proponent should be interested in what would happen to the patients in groups 8 and 9.

Human convalescent poliomyelitis serum has been recommended for treatment in the acute stage, despite the fact that at no time has there been any evidence that is was of value. It could not be shown that the patient with simple somatic peripheral paralysis was benefited. The mortality rate of bulbar poliomyelitis in

some epidemics may be as high as 75 per cent. Patients with bulbar poliomyelitis have been treated with serum, and it has been stated that convalescent serum must be of some value, since the mortality of these treated patients was 30 per cent. There were no control patients. This conclusion appeared logical and was accepted in some quarters as almost positive proof that serum did some good. Yet at the same time, in another city, during the same epidemic, there had also been an increase in the number of cases of bulbar poliomyelitis and a decrease in the mortality rate to 28.5 per cent; on investigation, a similar situation has been found in other localities.

One can give a second example of the fallacy of statistics. Suppose, for example, one has 100 patients with infantile paralysis, 20 in groups 8 and 9 and 80 in groups 2 to 7 inclusive. It is known that 80 will get well no matter what is done. If the entire group of 100 is treated, the recovery rate is 80 per cent to start with, a fictitious rate to attribute to any therapy. In accepting rates of recovery, one should examine the type of patient the author treats. It may be found that the author who states that he had a recovery rate of 30 per cent for patients with paresis and paralysis (groups 8 and 9) actually has a better recovery rate than one who claims an 80 per cent recovery rate and who includes persons with all types of the disease.

Excuses for Failure.—Any system of therapy should be such that properly trained and intelligent persons can apply the principles advanced and obtain results similar to those of the advocate. It is true that some one particularly interested in a method of procedure will probably have incentive enough to see that the procedure is given more than a fair trial. On the other hand, one who has come to believe in a certain principle and has received training along that line should also do well. It is therefore unfortunate if the proponent of a system insists that only he or she can practice it efficiently and decries the efforts of his disciples when these efforts are not always followed by the desired results. It is noticeable that in the difficult cases the proponent of a therapy has a "way out" in the sense that he can claim that he was not actually in charge of the case—that if he had been the outcome would have been different. This attitude, of course, always tends to disturb the minds of the troubled parents, who may believe this silly innuendo. No therapy is 100 per cent perfect, and every physician will have therapeutic failures.

CONCLUSION

Enough has been said to indicate that poliomyelitis is a disease with protean manifestations and that these manifestations must be determined and made clear in order to evaluate the efficiency of any therapy.

News and Comment

GENERAL NEWS

Directory of Medical Specialists.—Announcement is made that the "Directory of Medical Specialists" is now to be published by the A. N. Marquis Company, of Chicago, publishers of "Who's Who in America." Previous editions have been published for the Advisory Board for Medical Specialties by the Columbia University Press, New York.

It is planned not to issue the next edition before 1945, on account of the war, but the A. N. Marquis Company will publish a supplemental list of all those who have been certified by the American boards since the last (1942) edition of the directory, totaling about 3,600. This is to be distributed at cost, and monthly or bimonthly bulletins listing successful candidates for certification at examinations during the interim before the next edition are to be issued as a subscribers' service.

Dr. Paul Titus (Pittsburgh), of the American Board of Obstetrics and Gynecology, will continue as the directing editor, and Dr. J. Stewart Rodman (Philadelphia), of the American Board of Surgery, as associate editor. The editorial board will be composed, as before, of the secretaries of the fifteen American boards.

Communications should be addressed to the Directing Editor, Directory of Medical Specialists, 919 North Michigan Avenue, Chicago (11).

Benjamin Knox Rachford Lectureships.—Dr. William E. Ladd, of Harvard University, will deliver the twelfth annual series of the Benjamin Knox Rachford Lectureships on Tuesday evening, Jan. 18, and on Wednesday evening, Jan. 19, 1944, at 8.15, in the auditorium of the Children's Hospital Clinic and Research Building, in Cincinnati. The subjects of his lectures will be "Surgical Manifestations of Congenital Malformations of the Alimentary Tract" and "Experiences with Esophageal Atresia and Tracheoesophageal Fistulae."

Scientific Exhibit of American Medical Association.—Those having material which they wish to present in the Scientific Exhibit at the meeting of the American Medical Association June 12 to 16, 1944 should communicate with Dr. Sterling H. Ashmun, 117 South Main Street, Dayton 2, Ohio.

Abstracts from Current Literature

Biochemistry; Bacteriology, and Pathology

STUDIES ON STAPHYLOCOCCI: I. OCCURRENCE OF BACTERIOPHAGE CARRIERS AMONG STRAINS OF STAPHYLOCOCCUS AUREUS. ROY T. FISK, J. Infect. Dis. **71**:153 (Sept.-Oct.) 1942.

Bacteriophage carriers are not uncommon among normal strains of Staphylococcus aureus. The incidence of lysogens was 44.2 per cent in this study with 43 strains.

Lysogens could not be demonstrated among cultures lacking the characteristics of pathogenic Staph. aureus, and these strains were resistant to phages isolated from lysogenic staphylococci.

The phages carried by staphylococci exhibit selective activity when tested on strains of the aureus group. In this manner twenty-four different phages were identified.

TOOMEY, Cleveland.

STUDIES ON STAPHYLOCOCCI: II. IDENTIFICATION OF STAPHYLOCOCCUS AUREUS STRAINS BY MEANS OF BACTERIOPHAGE. ROY T. FISK, J. Infect. Dis. **71**:161 (Sept.-Oct.) 1942.

The phage susceptibility of 95 culture strains of Staphylococcus aureus was determined with 27 different bacteriophages, the differential action of which made it possible to divide cultures of pathogenic staphylococci into groups.

Strains isolated from related sources were found to react to the same phages and could be differentiated from other cultures by this method.

TOOMEY, Cleveland.

RELATIONSHIP OF THE ST. LOUIS AND THE WESTERN EQUINE ENCEPHALITIC VIRUSES TO FOWL AND MAMMALS IN CALIFORNIA. BEATRICE F. HOWITT and WILLIAM VAN HERRICK, J. Infect. Dis. **71**:179 (Sept.-Oct.) 1942.

Antibodies against the St. Louis encephalitic virus have been found in the serums of normal fowl and of horses and other mammals, either alone or in combination with those of the western equine strain. A higher percentage of positive neutralization reactions for both of these viruses has been obtained from serums of apparently normal domestic fowl and mammals in endemic areas of California than in supposedly nonendemic regions, although antibody-containing serums may be found in animals in the latter districts, mainly in horses. It has also been shown that while the percentage of positive reactions was higher among domestic animals, a small number of wild rodents may harbor antibodies to the western equine and the St. Louis strain. Although neither the western equine nor the St. Louis virus has been recovered as yet from any of the rodent tissues, the virus of lymphocytic choriomeningitis has been obtained from the brain and organs of 2 wild house mice (Mus musculus).

TOOMEY, Cleveland.

CHARACTERISTICS OF THE TOXOPLASMA NEUTRALIZING ANTIBODY. ALBERT B. SABIN and ISAAC RUCHMAN, Proc. Soc. Exper. Biol. & Med. **51**:1 (Oct.) 1942.

Because of the fact that Toxoplasma is responsible for a variety of manifestations in human beings, the authors believed it would be useful to study certain serologic reactions associated with this protozoon parasite. Rhesus monkeys were bled before and after intracutaneous and intra-abdominal inoculations, and the serums were tested by injection into the backs of rabbits. Toxoplasma-neutralizing antibody could not be stored at 5 C. for one to two weeks and was destroyed at 56 C. in thirty minutes, but it could be preserved for months when kept in an insulated box frozen with solid carbon dioxide. Fresh normal monkey serum was devoid of neutralizing effect, but the antibody developed within one to two weeks after infection and persisted for months. The persistency was found not to be due to active infection with Toxoplasma, as was demonstrated by histologic examination of the tissues and by intracerebral and intra-abdominal inoculation into mice. Neutralizing antibodies were not demonstrable in fresh serums of rabbits, mice, cats or dogs after experimental infections with this parasitic organism. The neutralizing antibody in the serum obtained from an 18 month old infant with congenital toxoplasmosis was found to be labile to freezing temperature and storage.

HANSEN, Minneapolis.

TOXOPLASMA NEUTRALIZING ANTIBODY IN HUMAN BEINGS AND MORBID CONDITIONS ASSOCIATED WITH IT. ALBERT B. SABIN, Proc. Soc. Exper. Biol. & Med. **51**:6 (Oct.) 1942.

Because of the fact that Toxoplasma organisms have been found to be responsible for congenital encephalomyelitis and acute encephalitis in childhood and a syndrome resembling

spotted fever associated with pneumonitis, the author studied the effect of human serum for Toxoplasma-neutralizing antibodies by means of a rabbit skin neutralization test. Naturally nonimmune rabbits were inoculated with various dilutions of serum and Tyrode's solution (as controls) as well as with a suspension of R. H. strain of Toxoplasma obtained from mice which had been inoculated with Toxoplasma obtained from a human subject with fatal encephalitis. The reaction in the rabbit to Toxoplasma begins with an erythema on the fourth day; necrosis develops on the seventh or eighth day, and the animal dies usually between the ninth and the twelfth day. The serums of 151 subjects were selected because of the presence of the clinical tetrad of Toxoplasma encephalitis: ([*a*] hydrocephalus or microcephaly; [*b*] cerebral calcification; [*c*] chorioretinitis, and [*d*] disturbance in nervous function) or because of various symptoms, such as stillbirths, anencephalic monsters, hydrocephalus or encephalitis, in various members of the family. The serums of 59 of these selected subjects contained Toxoplasma. The highest percentage occurred in the infants and children having psychomotor disturbances with or without hydrocephalus or microcephaly when associated with cerebral calcification or chorioretinitis or both. The congenital character of the disease was confirmed by the regular presence of these antibodies in the mothers of the affected children. There was a high incidence of positive reactions (in 9 of 10 tests) among older children and adults presenting a chorioretinitis of unknown cause. Serum which was able to neutralize Toxoplasma was also found in a number of persons who had had a history of encephalitis.

HANSEN, Minneapolis.

THE COMPLEMENT FIXATION REACTION IN TOXOPLASMIC INFECTION. JOEL WARREN and ALBERT B. SABIN, Proc. Soc. Exper. Biol. & Med. **51**:11 (Oct.) 1942.

The authors were able to demonstrate in a frozen and thawed extract prepared from rabbit brain infected with Toxoplasma the presence of an antigen which gave a specific complement fixation with immune rhesus monkey serum. The serums of rabbits, dogs and cats were found to have no such reaction. The serums of 7 monkeys which had been infected by the intracutaneous and intra-abdominal routes revealed the presence of specific complement-fixing antibodies within one to four weeks after inoculation, but the antibodies disappeared as early as two months after inoculation in most instances. Sixty-three specimens of human serum from 56 subjects showed no reaction to rabbit brain antigen. The serums of 10 persons with various pathologic conditions which probably resulted from previous infection with Toxoplasma were able to fix the complement. These may have represented relatively recent infection, as the serums which contained neutralizing antibodies for Toxoplasma had no complement-fixing power.

HANSEN, Minneapolis.

THERAPEUTIC EFFECTIVENESS OF CERTAIN SULFONAMIDES ON INFECTION BY AN INTRACELLULAR PROTOZOON (TOXOPLASMA). ALBERT B. SABIN and JOEL WARREN, Proc. Soc. Exper. Biol. & Med. **51**:19 (Oct.) 1942.

Sulfathiazole and sulfapyridine when given as 1 per cent of the diet had an inhibitory action and curative effect on Toxoplasma infection in mice, the survival being greatly increased. Sulfanilamide had no such effect.

HANSEN, Minneapolis.

Metabolsim; Infant Feeding; Milk and Other Foods

THE AMOUNT OF FAT IN THE BLOOD AFTER A MEAL AS ESTIMATED BY COUNTING THE CHYLOMICRONS. RALPH R. COOPER and HARRY LUSK, Am. J. Digest. Dis. **9**:395 (Nov.) 1942.

A method of counting chylomicrons in the capillary blood is described. Counts are made on fasting blood, one hour after a specified meal and then at successive half-hour intervals. In 12 normal subjects the maximum number, averaging 117 per field, was reached at two hours. Patients with ulcerative colitis, obstructive jaundice and vomiting of pregnancy and 1 patient thought to have pancreatitis showed both a slower rise and a definitely lower number of chylomicrons per field. Analysis of venous blood showed a parallel between the neutral fat content and the number of these particles.

LESLIE, Evanston, Ill.

THE INFLUENCE OF DIETARY FAT UPON THE EXCRETION OF UROBILIN. H. W. JOSEPHS, L. E. HOLT JR., H. C. TIDWELL and C. KAJDI, Bull. Johns Hopkins Hosp. **71**:84 (Aug.) 1942.

Normal infants fed high fat diets show an increased output of urobilin in the feces. Small quantities of free fatty acids and soaps cause a similar increase. The authors think absorption of increased amounts of soap on a high fat diet leads to increased hemolysis.

LYTTLE, New York.

CHEMICAL COMPOSITION OF TWENTY-TWO COMMON FOODS AND COMPARISON OF ANALYTICAL WITH CALCULATED VALUES OF DIETS. FRANCES COPE HUMMEL, MARION L. SHEPHERD, HARRY GALBRAITH, H. H. WILLIAMS and ICIE G. MACY, J. Nutrition **24**:41 (July) 1942.

Hummel and associates present several tables showing the analyses of twenty-two common foods for nitrogen, fat, energy, calcium, magnesium, sodium, potassium, phosphorus, chlorine, sulfur and iron. The figures indicate that individual samples of a given food vary from values reported in the standard tables. Milk is more constant in mineral content than fruits and vegetables.

FREDEEN, Kansas City, Mo.

THE INFLUENCE OF ALUMINUM SULFATE AND ALUMINUM HYDROXIDE UPON THE ABSORPTION OF DIETARY PHOSPHORUS BY THE RAT. HAROLD R. STREET, J. NUTRITION **24**:111 (Aug.) 1942.

When a soluble form of aluminum, aluminum sulfate, was added in amounts chemically equivalent to the phosphorus in the ration fed rats, Street found that essentially all of the phosphorus was rendered unavailable to the rat, owing to the complete precipitation of phosphorus in the intestinal tract. However, when aluminum hydroxide was added at levels of 0.5 and 1 per cent of the diet, only about one third or one fourth of the aluminum was converted to a form (aluminum chloride, soluble in acid gastric contents) reacting with the phosphorus.

FREDEEN, Kansas City, Mo.

SOFT CURD HOMOGENIZED MILK IN INFANT FEEDING. IRVING J. WOLMAN, SYDNEY BOROWSKY, RAE NICHOLAS and BERNHARD SPUHR, J. Pediat. **21**:45 (July) 1942.

Clinical investigation of soft curd homogenized milk as compared with boiled pasteurized milk was undertaken by the authors on 843 infants. Three varieties of homogenized milk were used, namely, sonic homogenized milk, low pressure homogenized milk and high pressure homogenized milk. The control pasteurized milk formulas were boiled for five minutes and then carbohydrate was added. The homogenized formulas were prepared in the original quart bottles at refrigerator temperatures, and the only alteration was the addition of carbohydrate. Suitable dietary additions (solid foods, vitamins, etc.) were administered to all children, depending on age. The undiluted, unboiled homogenized formulas were well taken by all children, and they thrived and gained. Gastrointestinal upsets, regurgitation, diarrhea, etc., were of low incidence and were no more frequent than in the control group fed pasteurized milk. Gain in weight and height was as good as in the control group. The homogenized formulas, particularly those subjected to temperatures of 150 to 160 F. for thirty minutes, had a low bacterial count, usually below 2,000 per cubic centimeter. Curd tension was also low.

Wolman suggests a threshold delimiting readily digested milks from those more poorly tolerated. On the safe, or proper, side of this threshold are the soft curd milks, and on the other side, the hard curd and less thoroughly processed milks.

STETTENBENZ, Buffalo.

BACTERIOLOGIC AND CHEMOTHERAPEUTIC STUDIES IN ACUTE DIARRHEA OF INFANTS AND CHILDREN. S. R. HALPERN and JOSEPHINE CUNNINGHAM, J. Pediat. **21**:184 (Aug.) 1942.

Sulfaguanidine and sulfathiazole produced uniformly good results in 36 cases of bacillary dysentery in which the diagnosis was confirmed by pus in the stools, by a positive bacteriologic culture and by a positive agglutination titer of 1:40 and higher. Stools became normal in five to six days. Several children who appeared moribund made dramatic recoveries. Other measures included parenteral administration of fluids, transfusions and diet. Sulfathiazole is of value from the fact that it can be given parenterally to a moribund patient.

STETTENBENZ, Buffalo, N. Y.

OBESITY IN CHILDREN. I. P. BRONSTEIN, L. J. HALPERN and A. W. BROWN, J. Pediat. **21**:485 (Oct.) 1942.

Forty-six obese children whose conditions were tentatively diagnosed as Fröhlich's syndrome were thoroughly studied by repeated laboratory and physical check-up. Definite evidence of any glandular dysfunction was not found in any child. Therapy consisted in restricted intake of food and fluids, exercise and administration of vitamins. Loss of weight occurred only if the child followed this regimen strictly, but for the most part cooperation was poor. Thyroid, pituitary extracts and chorionic gonadotropin had no effect in reducing weight. Amphetamine sulfate, while causing a reduction in weight through loss of appetite, was of only temporary value. The authors believe that most mentally normal obese children tend to regulate themselves at puberty as a matter of pride.

STETTENBENZ, Buffalo.

DETERIORATION OF MILK BY BACTERIAL GROWTH UNDER REFRIGERATION AT 40° F. FRANK E. MOTT and HENRY MAZER, New England J. Med. **227**:174 (July 30) 1942.

The original investigation included grade A pasteurized milk, commonly called "grade A milk," and grade B pasteurized milk, commonly called "milk-pasteurized," but later it was enlarged to include certified pasteurized milk, produced under the supervision of the Medical Milk Commission. The samples of milk were obtained from vehicles on the streets of Boston engaged in the delivery of milk.

After seventy-two hours in the refrigerator at 40 F., both grade A pasteurized milk and grade B pasteurized milk had such a growth of colonies of bacteria from 1 cc. that their sale would be in violation of the law. Certified pasteurized milk, on the other hand, even after ninety-six hours in the refrigerator, still conformed to the bacterial standards and was perfectly safe for use as measured by present knowledge of the subject. After four days in the refrigerator at 40 F., both grade A pasteurized milk and grade B pasteurized milk had bacterial contents so great that their use might reasonably be expected to cause diarrhea and enteritis in children under 6 years of age.

In view of these findings, it is evident that the present conception of the inhibiting effect of refrigeration on bacterial growth in milk must be revised. GENGENBACH, Denver.

ACCELERATED RAT GROWTH RATE ON DIETARY NITROGEN OBTAINED FROM PANCREAS. ABRAHAM WHITE and MARION A. SAYERS, Proc. Soc. Exper. Biol. & Med. **51**:270 (Nov.) 1942.

Purified pancreatic protein material obtained from beef pancreas prepared as reported by Kazal, Westfall, Ciereszko, Risley and Arnow (*Proc. Soc. Exper. Biol. & Med.* **51**:268, 1942) was fed at the 18 per cent level to male white rats of the Yale strain. The rate of growth and the amount of food consumed by the animals fed the pancreatic protein were superior to those of the rats given diets containing casein, pumpkin seed globulin, beef serum or soy beans as the source of protein. HANSEN, Minneapolis.

CONDITION OF RETAILED RAW MILK IN AN URBAN AREA. C. A. GREEN, Brit. M. J. **2**:9 (July 4) 1942.

The incidence of tuberculosis of the bovine type in human subjects is regrettably high in England and Scotland. The lesions of the bovine type in human subjects are usually the result of ingestion of milk from infected cattle. With these facts in mind, 116 samples of raw milk were examined for bacterial count and for the presence of tubercle bacilli. Between 40 and 50 per cent of these samples failed to come up to satisfactory standards of cleanliness. It was also found that 20 per cent of the milks tested yielded tubercle bacilli. From these results it can be seen that raw milk is definitely unsatisfactory for consumption, and it is interesting that in some of these milks whose gross bacterial counts was not high tubercle bacilli were found.

In his conclusions the author states that compulsory pasteurization of milk is considered an essential measure. BIRDSONG, University, Va.

DIETARY STANDARDS RECOMMENDED BY THE NATIONAL NUTRITION COUNCIL, DEPARTMENT OF PUBLIC HEALTH, FOR USE IN SOUTH AFRICA. South African M. J. **15**:291 (Aug. 8) 1942.

Recommended Daily Allowances for Specific Nutrients

	Calories	Protein, Gm. (1)	Calcium, Gm. (2)	Iron, Mg.	Vitamin A, International Units (3)	Vitamin B_1 (Thiamine) Mg. (4)	Vitamin C (Ascorbic Acid) Mg. (5)
Children (up to 12 years)							
Under 1 year.....	100 per Kg.	3-4 per Kg.	0.8*	6	450-750	(0.5)	30
1-3 years.....	1,200	40	(0.9)†	7	1,000	(0.5)	(35)
4-6 years.....	1,600	50	(0.9)	8	2,500	(0.5)	(50)
7-9 years.....	2,000	60	(0.9)	10	3,000	(1.0)	(50)
10-12 years.....	2,500	70	1.2	12	3,000	(1.0)	(50)
Children (over 12 years)							
Girls: 13-15 years....	2,800	60	1.3	15	4,000-5,000	(1.5)	(50)
16-20 years....	2,400	75	1.0	15	4,000-5,000	(1.5)	(50)
Boys: 13-15 years....	3,200	85	1.4	15	4,000-5,000	(2.0)	(50)
16-20 years....	3,800	100	1.4	15	4,000-5,000	(2.0)	(50)

* 6 months to 2 years.

† 2 to 3 years.

Figures in brackets are intended to serve as guides.

1. *Protein.*—In order to ensure protein quality, one third, or preferably one half, should be obtained from animal sources.

2. *Calcium.*—These figures may be regarded as at least adequate, under South African conditions; figures may require revision as a result of local investigations.

3. *Vitamin A.*—Tentative values; larger amounts may be necessary where the vitamin is largely derived from carotene.

4. *Vitamin B_1.*—These amounts may need to be increased.

5. *Vitamin C.*—Tentative values; requirements for various ages and conditions cannot yet be regarded as established.

6. *Vitamin D.*—No estimate has been made for this vitamin; an adequate supply is assumed, provided light clothing is worn and there is reasonable exposure to sunlight.

7. *Nicotinic Acid.*—A daily intake of 10 mg. of this vitamin is suggested for an adult.

8. *Riboflavin.*—Two milligrams of riboflavin is considered adequate for an adult.

GONCE, Madison, Wis.

DIAGRAM OF GLYCEMIA. CESAR SANTOS, Rev. méd. latino-am. **27**:49 (July) 1942.

Santos offers a schematic presentation of dextrose metabolism which contributes to an understanding of diabetes.

DWYER, Kansas City, Mo.

Vitamins; Avitaminoses

ACTION OF INTESTINAL MICROORGANISMS ON ASCORBIC ACID. RAYMOND M. YOUNG and L. H. JAMES, J. Bact. **44**:75 (July) 1942.

Extensive experiments were carried out by the authors to observe the effect of bacteria on ascorbic acid in the intestines. It was found that several direct cultures of human feces and isolated cultures of Escherichia coli and Aerobacter aerogenes were able to decompose vitamin C under both aerobic and anaerobic conditions. On the other hand, organisms of the Proteus and Alcaligenes groups failed to attack the vitamin and definitely protected the substance from atmospheric oxidation, but the protective action was not operative in the presence of vitamin C–decomposing bacteria. Furthermore, the presence of dextrose or lactose in ascorbic acid nutrient medium definitely spared the vitamin from oxidation by the fecal cultures and pure cultures of Escherichia coli. The sparing action was operative under aerobic and anaerobic conditions. The vitamin disappeared rapidly from the medium as soon as the sugar had been completely utilized.

STOESSER, Minneapolis.

THE RELATION BETWEEN DARK ADAPTATION AND THE LEVEL OF VITAMIN A IN THE BLOOD. CHARLES HAIG and ARTHUR J. PATEK JR., J. Clin. Investigation **21**:377 (July) 1942.

Simultaneous measurements of dark adaptation and the vitamin A and carotenoid levels of the plasma were obtained on normal persons and on persons with cirrhosis of the liver and other chronic diseases. The data thus obtained did not indicate a causal relation between the level of vitamin A in the blood and the rate and extent of dark adaptation.

GUEST, Cincinnati.

TISSUE THIAMIN CONCENTRATIONS AND URINARY THIAMIN EXCRETION. JOSEPH W. FERREBEE, NORMAN WEISSMAN, DONALD PARKER and PHILIP S. OWEN, J. Clin. Investigation **21**:401 (July) 1942.

The concentrations of thiamine found in human tissue obtained at autopsy were of the order of 2 to 3 micrograms per gram for heart muscle, 0.5 microgram per gram for skeletal muscle and 1 microgram per gram for brain, liver and kidney. The authors point out that concentrations significantly below these may be found in persons suffering prolonged febrile illnesses, with dietary restrictions, and that higher concentrations may be found in children and patients who have recently received large quantities of the vitamin. While deficient subjects tend to excrete less thiamine than normal subjects, such a tendency appears insufficient to permit the recognition of small changes in concentration in the tissues by measurements of the amount of thiamine excreted.

GUEST, Cincinnati.

THE MOBILIZATION BY ALCOHOLS OF VITAMIN A FROM ITS STORES IN THE TISSUES. S. W. CLAUSEN, W. S. BAUM, A. B. MCCOORD, J. O. RYDEEN and B. B. BREESE, J. Nutrition **24**:1 (July) 1942.

Clausen and co-workers found in their work with dogs a marked increase in the vitamin A concentration of the serum following administration of methyl, ethyl, propyl and isopropyl alcohols. The mode of administration did not affect the results. It was shown that vitamin A was mobilized from its storage places in the body, such as the liver and fat depots.

FREDEEN, Kansas City, Mo.

A STUDY OF THE ASCORBIC ACID REQUIREMENTS OF CHILDREN OF EARLY SCHOOL AGE. VIVIAN M. ROBERTS and LYDIA J. ROBERTS, J. Nutrition **24**:25 (July) 1942.

A daily intake of 65 mg. to 75 mg. of ascorbic acid was required by children of the early school age to promote saturation on the basis of 50 per cent excretion of the test dose in twenty-four hours. These amounts, according to Roberts and Roberts, were sufficient to maintain blood levels above 0.7 mg. per hundred cubic centimeters and allow for average "retention."

FREDEEN, Kansas City, Mo.

THE UTILIZATION OF CAROTENE AND VITAMIN A IN THE RAT. RAY TREICHLER, A. R. KEMMERER and G. S. FRAPS, J. Nutrition **24**:57 (July) 1942.

Treichler, Kemmerer and Fraps, using paired feeding technic and spectrographic analytic methods, made investigations on the utilization of vitamin A and carotene for storage in the liver in rats. The vitamin A in cod liver oil was found the most efficient for building up liver stores; carotene dissolved in cottonseed oil was 59 per cent as effective, and carotene in alfalfa leaf meal was 21 per cent as effective. During the two weeks following weaning, despite the fact that the rats were on a vitamin A–free diet, the spectrographic vitamin A content of the liver increased significantly. Yeast present in the diet contributed definitely to this increase.

FREDEEN, Kansas City, Mo.

POLYNEUROPATHY IN THIAMINE DEFICIENT RATS DELAYED BY ALCOHOL OR WHISKY. J. V. LOWRY, W. H. SEBRELL, F. S. DAFT and L. L. ASHBURN, J. Nutrition **24**:73 (July) 1942.

Lowry, Sebrell, Daft and Ashburn presented data showing that the ingestion of alcohol or whisky delays the onset of polyneuropathy in thiamine-deficient rats.

FREDEEN, Kansas City, Mo.

STORAGE OF PYRIDOXINE IN THE RAT. LEOPOLD R. CERECEDO and JOHN R. FOY, J. Nutrition **24**:93 (July) 1942.

Cerecedo and Foy presented data showing that rats are capable of storing pyridoxine. It had been observed that weanling rats allowed to feed on the stock diet for ten days before being put on a pyridoxine-free diet and thus increasing their weight over that of controls require a much longer period to acquire dermatitis. The more vitamin the rats obtained during this period, the longer were they able to resist the dermatitis. Their data suggest that the irregularity in the occurrence of dermatitis in rats deprived of pyridoxine, as reported by others, depends in part on the differences in the reserves of the vitamin present in young animals at the start of experimentation.

FREDEEN, Kansas City, Mo.

THE ASCORBIC ACID CONTENT OF EWES' BLOOD, COLOSTRUM AND MILK AND THE EFFECT OF ASCORBIC ACID INJECTIONS. G. HOWARD SATTERFIELD, E. A. BAILEY JR., J. E. FOSTER and E. H. HOSTETLER, J. Nutrition **24**:121 (Aug.) 1942.

Satterfield and his co-workers found that the ascorbic acid content of ewes' blood is 0.43 to 0.82 mg. per hundred cubic centimeters of plasma and the content of colostrum 2.01 to 9.94 mg. per hundred cubic centimeters, dropping rapidly within five or six days to the constant value of 0.8 mg. per hundred cubic centimeters for the milk.

Injection of ascorbic acid resulted in a rise in the ascorbic acid level of both the blood and the milk, the blood returning to normal in one day and the milk in three days.

FREDEEN, Kansas City, Mo.

THIAMINE CLEARANCE AS AN INDEX OF NUTRITIONAL STATUS. DANIEL MELNICK and HENRY FIELD JR., J. Nutrition **24**:131 (Aug.) 1942.

In the present investigation Melnick and Field reported a simpler and more reliable procedure for the detection of thiamine deficiency which may be used routinely in clinical work.

The test is relatively simple, namely, the measurement of thiamine excreted in the urine four hours after the parenteral administration of 350 micrograms per square meter of body area. All normal subjects excreted in excess of 50 micrograms of total thiamine during the four-hour period following the injection.

FREDEEN, Kansas City, Mo.

VITAMIN B_1 (THIAMINE) REQUIREMENT OF MAN. DANIEL MELNICK, J. Nutrition **24**:139 (Aug.) 1942.

Melnick found that only 73 per cent of the so-called normal subjects who were not restricted as to choice of diet excreted sufficient quantities of thiamine in the urine to pass all the thiamine clearance tests. The suggested daily intake should be 500 micrograms per thousand calories; however, Melnick, on the basis of objective studies of thiamine balance, estimated the adult requirement to be 350 micrograms per thousand calories.

FREDEEN, Kansas City, Mo.

THE NICOTINIC ACID CONTENT OF MEAT. W. J. DANN and P. HANDLER, J. Nutrition **24**:153 (Aug.) 1942.

Dann and Handler present two tables showing the results of analyses for nicotinic acid of a number of meats purchased at retail stores. The values are considerably lower than those reported by others, as Dann and Handler based their observations on a chemical procedure using completely decolorized digests of the tissues. Their results are closely correlated with results obtained in a microbiologic assay. Generally 33 to 50 per cent of the nicotinic acid was lost through the various cooking procedures.

FREDEEN, Kansas City, Mo.

NICOTINIC ACID, PANTOTHENIC ACID AND PYRIDOXINE IN WHEAT AND WHEAT PRODUCTS. L. J. TEPLY, F. M. STRONG and C. A. ELVEHJEM, J. Nutrition **24**:167 (Aug.) 1942.

Patent flour, according to data presented by Teply, Strong and Elvehjem, contains approximately one sixth as much nicotinic acid as whole wheat and about one half as much pantothenic acid and pyridoxine.

FREDEEN, Kansas City, Mo.

THE RELATION OF B-VITAMINS AND DIETARY FAT TO THE LIPOTROPIC ACTION OF CHOLINE. R. W. ENGEL, J. Nutrition **24**:175 (Aug.) 1942.

Engel concluded that pyridoxine and a source of essential fatty acids are necessary in the diet of the rat for choline to function properly as a lipotropic agent. In addition to choline, for rats fed purified diets supplemented with the known B vitamins, inositol is a necessary dietary constituent.

FREDEEN, Kansas City, Mo.

CAROTINAEMA. S. ALMOND and R. F. L. LOGAN, Brit. M. J. **2**:239 (Aug. 29) 1942.

Five cases are reported in which a diagnosis of jaundice, pernicious anemia, hemolytic anemia and cholelithiasis had been made. In all of these cases it was found that the patients were suffering from carotenemia. In this series of cases the patients had been taking large amounts of carrots over a long period of time because of their abundance in "warring England" and because of the propaganda distributed on the value of the vitamin content of fresh vegetables. It was found that the pigment occurred most frequently in the nasal folds and on the palms of the hands, but it was also distributed throughout the skin. The main point in the differential diagnosis between carotenemia and jaundice in these patients was the relatively good health of the patients and the fact that the scleras remained clear of any yellow pigmentation. A simple three layer test by Greene and Blackford is described, in which serum, alcohol and purified petroleum benzine (petroleum ether) are mixed together and shaken. If the pigment is the lipochrome of carrots, the pigments will be absorbed by the petroleum ether. If the pigmentation is the result of abnormal concentration of bilirubin the pigment will be seen in the alcohol layer. In this study it was remarkably consistent that the patients had to be fed large amounts of carrots for about six or seven months before pigmentation became obvious. In one of the cases reported, the patient was a newborn baby that was breast fed. Its mother manifested carotenemia, and after about two months of breast feeding the child acquired a definite carotenemia. This was believed to be the first reported case of carotenemia from the breast milk of an infected mother.

BIRDSONG, University, Va.

Hygiene; Growth and Nutrition; Public Health

MASSACHUSETTS PUBLIC HEALTH PROBLEMS IN WARTIME. PAUL J. JAKMAUH, New England J. Med. **227**:206 (Aug. 6) 1942.

This article should be read by all physicians interested in problems of public health, both in individual states and in individual communities, but certain points may be emphasized.

After a critical review of the high percentage of rejections for physical defects among young men examined under the Selective Service Act, there came a realization of the need for increased health service for the youth of the nation. Although it is true that many of the defects date back to inadequacies which had their origin during infancy and early childhood, it is evident that increasing emphasis must be placed on the preservation of health during later childhood and adolescence.

The increase in tuberculosis among young women already noted in England is a warning that should not be allowed to go unheeded. Each district health officer should make every effort to apprise himself of the health needs and resources of the various communities in his district and should endeavor, through the efficient use of his staff, to find a solution for these needs.

Responsibility for the work of these units in Massachusetts has been delegated to the assistant to the State Commissioner of Health as part of local health administration.

GENGENBACH, Denver.

THE FINDINGS OF THE CAPE NUTRITION SURVEY. J. F. BROCH and J. M. LATSKY, South African M. J. **16**:255 (July 11) 1942.

The state of nutrition among certain groups of Cape Peninsula school children between the ages of 8 and 16 years was assessed by the following examinations: physical examination, somatic measurements, complete blood count, sedimentation rate, Wassermann reaction, roentgen examination of one wrist, dental examination, Sulkowitch test for urinary calcium, biophotometric test and vitamin C saturation test. As a result, it was found that fewer than 43 per cent of the Cape colored children of the peninsula are in normal health and of normal nutrition, and in certain districts the situation among European school children is almost as bad. The available evidence suggests that the main factor in this malnutrition is a general deficiency of protective foods.

GONCE, Madison, Wis.

IMPORTANCE OF INTRODUCING INTO THE SCIENTIFIC LEXICON THE TERM PAIDOFILAXIS OR PRESERVATION OF CHILDHOOD. LUIS FELIPE GONZÁLEZ, Bol. Inst. internac. am. de protec. a la infancia **16**:82 (July) 1942.

It would be desirable to introduce into the scientific lexicon the term *paidofilaxis* [pedophylaxis] as a name for the new discipline in connection with child welfare problems.

The aims of pedophylaxis should be (*a*) the preservation of childhood from the evils that surround it and (*b*) the study and application of the scientific principles destined to assure the best conditions for the gestation and birth of the child, as well as for his complete development until the moment when he is capable of looking after himself and of being an active element in the society of which he forms a part.

[EDITOR'S SUMMARY]

IMPORTANCE OF THE TEACHING OF PAIDOFILAXIS IN NORMAL SCHOOLS AND SCHOOLS OF SOCIAL SERVICE. DUIS FELIPE GONZÁLEZ, Bol. Inst. internac. am. de protec. a la infancia **16**:87 (July) 1942.

Before the Eighth Panamerican Child Congress the author recommended that the teaching of *paidofilaxis*, a scientific activity for the study and solution of childhood problems, should be adopted by the normal schools and by the schools of social service as a subject in the curriculum of the teaching centers.

[EDITOR'S SUMMARY]

Prematurity and Congenital Deformities

FETAL SURVIVAL FOLLOWING THE INJECTION OF ANTUITRIN-S IN PREGNANT RATS AND RABBITS. R. M. COCO, Am. J. Physiol. **137**:143 (Aug.) 1942.

Coco found that varying amounts of chorionic gonadotropin injected into pregnant rats and rabbits in late pregnancy usually resulted in death of the fetus in utero or soon after parturition and in rare instances in postponed and prolonged parturition. The ovaries usually contained induced corpora lutea, but these evidently did not reach a threshold level of secretion.

Since there was a high mortality rate of fetuses in utero before or at term and a low incidence of living, postmature fetuses, it appears that the chorionic gonadotropin acted as an abortient rather than as an agent prolonging gestation. NOURSE, Cleveland.

COMPLETE SITUS INVERSUS. JOSEPH G. PASTERNACK, New England J. Med. **227**:953 (Dec. 17) 1942.

The treatment and conditions observed at autopsy in a case of complete situs inversus associated with calcareous aortic stenosis, cor bovinum and multiple pulmonary infarcts are reported for the first time. The cardiac lesions were compatible with an old rheumatic infection and occurred in a man of 43 years. GENGENBACH, Denver.

CYCLOPS AND ANENCEPHALUS. CID FERREIRA JORGE, Rev. méd. munic. **4**:57 (July) 1942.

The author describes and shows photographs of 3 fetal monsters, 1 cyclops and 2 anencephalous monsters. He discusses various causative factors and believes that syphilis was the effective factor in these instances of teratism. SANFORD, Chicago.

Newborn

INTESTINAL OBSTRUCTION IN THE NEWBORN. J. W. DUCKETT, Ann. Surg. **116**:321 (Sept.) 1942.

Surgical treatment of congenital obstruction of the intestine in the newborn is at last beginning to produce encouraging results. Thirty years ago not a single successful operation had been announced.

Diagnosis can often be made on the history alone. In the absence of intracranial birth injury or unusual infection, bile-stained projectile vomiting beginning soon after birth and persisting almost invariably means obstruction of the intestine. Positive diagnostic aid may often be obtained from roentgenologic studies. Plain roentgenograms of the abdomen are usually adequate.

Survival of the infant with complete obstruction depends on early diagnosis and immediate repair of the defect. Without operation death usually occurs in seven to ten days. Preoperative care requires administration of vitamin K to jaundiced babies and giving of fluids and blood to dehydrated babies. Ether anesthesia is preferable.

Six case reports follow. BANTIN, Omaha.

SKELETAL CHANGES ASSOCIATED WITH ERYTHROBLASTOSIS FETALIS. RICHARD H. FOLLIS JR., DEBORAH JACKSON and WILLIAM H. CARNES, J. Pediat. **21**:80 (July) 1942.

Follis, Jackson and Carnes describe the osseous changes by roentgen and by microscopic examination in 5 cases of erythroblastosis fetalis. Consistent increased density of the shafts of the bones was found in all the cases and bands of decreased density, particularly at the ends of bones in 3. STETTENBENZ, Buffalo.

EFFECTS OF ANOXIA AT BIRTH ON CENTRAL NERVOUS SYSTEM OF THE GUINEA PIG. W. F. WINDLE and R. F. BECKER, Proc. Soc. Exper. Biol. & Med. **51**:213 (Nov.) 1942.

Extensive studies of the effect of anoxia on the development of guinea pigs were undertaken on 103 animals by inducing anoxia of varying degrees at the time of birth. Evidence of damage to the central nervous system, such as motor weakness, tremors and inability to get up on the feet, were observed early, and when anoxia lasted over eight minutes such symptoms as decerebrate states, marked ataxia, convulsions, paralysis, somnolence, hyperesthesia and hypoesthesia and changes in behavior resulted. Various degrees of histologic changes were noted in the brain and cord. The authors conclude that neonatal asphyxia (asphyxia pallida) may induce irreparable fortuitous destruction of large or small regions of the brain.

HANSEN, Minneapolis.

HAEMOGLOBIN VALUES IN THE BLOOD OF NEWBORN INFANTS. J. D. HICKS, M. J. Australia **2**:117 (Aug. 15) 1942.

The range of hemoglobin content of the blood of full term babies within the first twelve hours of life was from 115 per cent (16.1 Gm.) to 201 per cent (27.2 Gm.), with a mean of 159.36 per cent (22.3 Gm.). The numbers of erythrocytes per cubic millimeter ranged from 4,650,000 to 9,670,000, with a mean figure of 6,950,000. The minimum number of leukocytes was 9,100 per cubic millimeter; the maximum was 47,350, and the mean was 22,500. There

was no significant relation between hemoglobin value and birth weight. The author offers the suggestion (not conclusion) that although a moderate anemia in the mother induces a polycythemia in the fetus a severe anemia in the mother is followed by anemia in the child.

GONCE, Madison, Wis.

SULPHAEMOGLOBIN(CYTH)AEMIA AND ANAEMIA NEONATORUM. A. WHITE FRANKLIN, Proc. Roy. Soc. Med. **25**:686 (Aug.) 1942.

Franklin reports the case of a baby, a first child, who had been delivered by forceps, which resulted in palsy of the left side of the face. Breathing was bubbly for two days, but feeding progress was normal until the seventh day, when he vomited bright green curds during the night. Next morning the skin was green and the lips blue. The liver and spleen were not enlarged. No drugs had been given to mother or baby. Vomiting continued sporadically, and the stools were dark green. The blood appeared dark brown, and sulfhemoglobin was found in a concentration of about 8 per cent. It was intracorpuscular, and Schumm's test on the plasma gave a negative reaction. A few days later the skin was less green and the cyanosis was less pronounced but the mucous membranes were pale. The child appeared severely anemic, the skin being tinged faintly green. The urine contained much urobilin only. Stool cultures yielded no abnormal bacteria. Transfusions of citrated blood were given. In this case intracorpuscular sulfhemoglobin apparently developed spontaneously on the ninth day of life and was followed by an acute anemia which responded to transfusions. At no time was there clinical jaundice, nor was there ever erythroblastosis. Blood counts before and after the transfusions are given in the report. The baby did not thrive. Death occurred at 7 weeks from bronchopneumonia and multiple abscesses of the lungs.

WILLIAMSON, New Orleans.

TETANUS IN THE NEWBORN. ORTIZ MACHEDO, Arch. de med. inf. **11**:127 (July-Sept.) 1942.

A newborn infant was infected with tetanus virus through the umbilical cord. Recovery followed after twenty-eight days. The author also reports the case of another infant of 3 months who was infected medically with this virus and recovered. Little antitetanus serum was given; more reliance was placed on chloral hydrate and calcium chloride.

[illegible]NFORD, Chicago.

NEONATAL MORTALITY IN ASUNCIÓN. RICARDO ODRIOSOLA, Bol. Inst. [illegible]ac. am. de protec. a la infancia **16**:91 (July) 1942.

The author refers to the attention given by investigators to early mortality as an important factor in infant mortality; he recalls Westgard's chart about mortality in Berlin and also refers to the table of vital statistics for Caracas from 1937 to 1939.

In relation to the charts published at the end of his work, he states that in Asunción during twenty-five years (1916 to 1940) 3,855 children died during the first month—an infant mortality from 28.9 to 44 per cent of the general mortality.

Studying the causes of mortality during the first month, he arrived at the conclusion that three diseases are largely responsible: infantile tetanus, 28.8 per cent; congenital weakness, 18.1 per cent; hereditary syphilis, 17.9 per cent. [FROM THE EDITOR'S SUMMARY.]

Acute Contagious Diseases

TREATMENT, COMPLICATIONS AND DEATHS IN 753 CASES OF CLINICAL DIPHTHERIA. YOSHIYE TOGASAKI, LEON ROSOVE, ALBERT C. BOWER and PAUL M. HAMILTON, Am. J. M. Sc. **204**:218 (Aug.) 1942.

The authors studied the cases of 753 patients with diphtheria admitted to the Los Angeles County General Hospital between July 1, 1937 and June 30, 1940. Thirty-six died. Forty-eight per cent were males, and 52 per cent were females. Sixty-one per cent were under 10 years of age. The average duration of illness before antitoxin was given was three and seven-tenths days. Greater duration was shown to increase the hazard.

The dosage of antitoxin was 20,000, 30,000 and 40,000 units for each of three groups, each containing 28 per cent of the patients, with lesser and larger amounts given intramuscularly to a few patients except those whose condition was extremely toxic.

Therapy as used in the Communicable Disease Unit of the Los Angeles County General Hospital is discussed.

Complications of myocarditis, bronchopneumonia, asphyxia and stenosis and other miscellaneous conditions are considered and treatment outlined.

Of 36 deaths, 22 were in cases in which tracheotomy was required. Twenty of the patients who died were of the "bull neck" type.

HENSKE, Omaha.

CONCURRENT IMMUNIZATION AGAINST TETANUS, DIPHTHERIA, AND PERTUSSIS. JOHN J. MILLER JR. and T. M. SAITO, J. Pediat. **21**:31 (July) 1942.

Miller and Saito report excellent results in immunization from the use of alum-precipitated tetanus and diphtheria toxoids concurrently with Haemophilus pertussis vaccine. Fluid toxoids were also given.

It was interesting that tetanus antitoxin levels were definitely lower when fluid toxoids were given, although satisfactory immunity was obtained to diphtheria and pertussis. Pertussis agglutinins were higher when vaccine was combined with alum-precipitated toxoids than when fluid toxoids were used or when pertussis vaccine was used alone. Miller and Saito believe that tetanus antitoxin levels are higher when the toxoid is combined with diphtheria toxoid than when tetanus toxoid alone is used. They recommend a three month interval between the first and the second injection of combined alum-precipitated toxoids. A third dose of tetanus toxoid alone is given three to four months later. STETTENBENZ, Buffalo.

A STUDY OF THE "SKIN TEST" WITH MENINGOCOCCUS TOXINS IN A GROUP OF BOYS. A. P. HITCHENS, S. E. BRANHAM and M. B. ROOT, Pub. Health Rep. **57**:1015 (July 10) 1942.

A group of 492 boys were tested intradermally at least once, 130 at least twice and 42 three or four times with Ferry's toxin (group I and group II toxins). Over one-half the total number of boys tested had positive reactions (a larger number to group II toxin and then to group I). Fifty-two of these were retested six months later with similar results, but there were an almost equal number (15) of shifts of reaction from positive to negative and from negative to positive. No explanation is offered, but the possibility of a seasonal variation in cutaneous reactivity is suggested. Until an outbreak of meningitis occurs the value of cutaneous tests cannot be appraised. SANFORD, Chicago.

STUDIES ON BACTERICIDAL AND PHAGOCYTIC ACTIVITY OF NORMAL BLOOD ON MENINGOCOCCI IN RELATION TO THE "SKIN TEST" WITH MENINGOCOCCUS TOXINS. S. E. BRANHAM, A. P. HITCHENS and M. B. ROOT, Pub. Health Rep. **57**:1021 (July 10) 1942.

The bactericidal activity of the blood of 50 boys from the same group who had been given cutaneous tests was studied. At least one half of all boys tested gave a positive cutaneous reaction to meningococcus toxins. The blood of about one half of the boys showed pronounced bactericidal activity for meningococci. Phagocytosis of meningococci by the leukocytes in blood from normal boys was never pronounced and varied from 0 to 32 per cent. There was no correlation between the three phenomena studied. Individual strains of meningococci varied greatly in their response to bactericidal and phagocytic activity. SANFORD, Chicago.

TRANSMISSION OF RUBELLA TO MACACUS MULATTA MONKEYS. KARL HABEL, Pub. Health Rep. **57**:1126 (July 31) 1942.

Macacus mulatta monkeys are susceptible to virus-containing material introduced intranasally, subcutaneously, intraperitoneally and intravenously. Viable virus was demonstrated in all patients within twelve hours after the onset of the rash. One patient was bled on the third day, and the monkey did not contract rubella. The same was true of nasal washings. Culture of the infective agent of rubella on the chorioallantoic membrane of developing chick embryos was successful in two of four attempts. It is suggested that immunity following rubella may be slow in developing. SANFORD, Chicago.

DIPHTHERIA ON THE WITWATERSRAND: A BACTERIOLOGICAL, CLINICAL AND EPIDEMIOLOGICAL SURVEY. J. F. MURRAY, South African M. J. **16**:247 (July 11) 1942.

Four hundred and seventy-five strains of Corynebacterium diphtheriae included 87.5 per cent mitis strains, 0.4 per cent intermediate, 4.2 per cent gravis and 7.6 per cent atypical. Forty-five per cent of the 20 gravis strains were avirulent. The case fatality rate in 246 mitis infections was 9.3 per cent, this high rate mostly being due to late culturing. Laryngeal involvement occurred in 37 cases (11.3 per cent); the infecting strain in 36 of these cases was of the mitis type. Of 197 normal young adults 136 (69 per cent) had positive Schick reactions. From 462 normal school children no virulent and only 15 avirulent strains were recovered. GONCE, Madison, Wis.

Acute Infectious Diseases

SALMONELLA INFECTION IN INFANTS AND CHILDREN. SIEGBERT BORNSTEIN and HERMAN SCHWARTZ, Am. J. M. Sc. **204**:546 (Oct.) 1942.

In a study of 369 cases of human Salmonella infections in North America and in Cuba in which clinical information was available the types of the organisms were classified by the authors at the New York Salmonella Center connected with the Beth Israel Hospital. Fifty cultures came from infants and young children. The clinical picture was that of septic infection in 9 subjects and in the remaining 41 children the predominant symptom was diarrhea. The authors reported 8 cases of Salmonella infections observed in the pediatric department of the hospital between February 1939 and October 1941. There were 3 cases of simple gastroenteritis due to Salmonella typhi murium involving infants of 3 and 5 months and a child of 3 years. A 10 year old boy with typhoid due to Salmonella paratyphi B received sulfaguanidine for forty days, but [illegible] stools were not freed from the organisms. One subject had a purulent lesion; 1 had typhoid due to S. typhi murium, and 2 infants had diarrhea due to more unusual types of Salmonella, namely Salmonella newport and Salmonella montevideo. In the authors' experience S. typhi murium is the most common type found in human beings and in 9 of 10 cases causes a rather harmless gastroenteritis.

The authors recommend routine cultural examination of a series of stool specimens from all infants and children with gastrointestinal symptoms. In all doubtful infections of more than a week's duration, agglutination tests with a variety of Salmonella antigens should be made. The blood should also be cultured in cases of suspected typhoid or of generalized septic infection.

HENSKE, Omaha.

FIRST RECORD OF A CASE OF HUMAN INFECTION WITH TAPEWORMS OF THE GENUS MESOCESTOIDES. ASA C. CHANDLER, Am. J. Trop. Med. **22**:493 (Sept.) 1942.

The author reports a case of infection with a tapeworm of the genus Mesocestoides occurring in a white infant 13 months of age. A detailed description of species infecting human beings and of the eggs of these worms is given. The life cycle of Mesocestoides has not been completely elucidated as yet. Human infection is thought to occur from eating improperly cooked flesh of wild mammals infected with this type of tapeworm.

BURPEE, Augusta, Ga.

RELATION OF HOOKWORM TO PHYSICAL STATUS IN GEORGIA. ARTHUR W. HILL and JUSTIN ANDREWS, Am. J. Trop. Med. **22**:499 (Sept.) 1942.

The authors studied white persons under 20 years of age in four counties in the southern part of Georgia. They considered the relation of hemoglobin values to egg counts and also the relation of hemoglobin values, heights and weights to egg counts and of physical status to egg counts and clinical changes in advanced stages of infection.

They concluded that the symptoms of hookworm infection are essentially those of progressive secondary anemia. Inadequate diet and resulting malnutrition augment the anemia due to hookworms. Definite clinical hookworm disease in southern Georgia is found in persons whose egg count averages above 5,000 eggs per cubic centimeter. Symptoms of hookworm disease can be temporarily relieved by the use of iron therapy. More rapid and permanent results can be obtained by the combined use of anthelmintics, iron therapy and improved diets.

BURPEE, Augusta, Ga.

SALICYLATE PROPHYLAXIS IN RHEUMATIC FEVER. ALVIN F. COBURN and LUCILE V. MOORE, J. Pediat. **21**:180 (Aug.) 1942.

Coburn and Moore administered 4 to 6 Gm. of sodium salicylate daily for four weeks to 47 young patients with quiescent rheumatic fever. Administration of the drug was started at the onset of symptoms of pharyngitis. Only 1 patient showed clinical manifestations of acute rheumatic fever. Of 139 control subjects, rheumatic fever developed in 57.

STETTENBENZ, Buffalo, N. Y.

RELATIVE IN VITRO EFFECTS OF THE SULFONAMIDES ON HEMOPHILUS INFLUENZAE. MILAN NOVAK and ANNA MARGARET LACY, J. Pediat. **21**:321 (Sept.) 1942.

A study of the effect of sulfanilamide, sulfapyridine, sulfadiazine and sulfathiazole on the growth of cultures of five strains of Haemophilus influenzae was made. In vitro sulfanilamide was relatively ineffective in inhibiting growth of H. influenzae; the other drugs readily inhibited the growth in concentrations as low as 2.5 to 5 mg. per hundred cubic centimeters. Clinically, also, results have been more successful with sulfapyridine, sulfadiazine and sulfathiazole; results with sulfanilamide have been disappointing.

STETTENBENZ, Buffalo, N. Y.

THE TRANSMISSION OF ENDAMEBA HISTOLYTICA AND AMEBIC DISEASE. J. ANDREWS, South. M. J. **35**:693 (July) 1942.

The author states that persons who are reservoirs of amebic infection are everywhere. They probably constitute from 5 to 10 per cent of the population, being most numerous in the South and least numerous in the northeastern region of this country. Transmission occurs most frequently by means of polluted water supplies, insanitary disposal of excreta, personal contact with carriers, insect conveyance and contaminated foods. Epidemic transmission may be due to water or to flies.

Factors that may be concerned in the production or suppression of symptoms appear to be temperature, strains of Endamoeba histolytica that are intrinsically more or less virulent than others, rapid passage from one host to another, dietary effects and immunity acquired by experience with the infection.

SCHLUTZ, Chicago.

THE CLINICAL ASPECTS OF AMEBIC DYSENTERY AS SEEN IN NORTH CAROLINA. W. SCHULZE and J. M. RUFFIN, South M. J. **35**:699 (July) 1942.

The authors state that amebic infections are common in North Carolina.

The diagnosis of amebic dysentery by proctoscopy is simple.

Amebic abscesses of the liver occur without previous diarrhea. Specific therapy is highly efficacious.

A dietary deficiency may be an important factor in causing infection and relapses.

SCHLUTZ, Chicago.

STUDIES ON THE VALUE OF X-RAY THERAPY IN EXPERIMENTAL GAS GANGRENE INFECTION. E. SINGER, M. J. Australia **2**:1 (July 4) 1942.

Singer reports that the roentgen treatment of mice infected intramuscularly with either Clostridium welchii or vibrion septique (Clostridium oedematis maligni), alone or combined with local application of sulfanilamide powder, was ineffectual.

GONCE, Madison, Wis.

THE NEW SULFANILAMIDE DERIVATIVES IN THE EFFICACIOUS TREATMENT OF BACILLARY DYSENTERY. FERNANDO ABENTE HAEDO and ARTIGAR RODRÍGUEZ DIVENCENZI, Bol. Inst. internac. am. de protec. a la infancia **16**:113 (July) 1942.

The authors fully confirm the efficacy of sulfonamide compounds in the treatment of bacillary dysentery. This treatment was started by them in October 1939.

The authors present observations (corroborated bacteriologically) on 16 new cases of bacillary dysentery belonging to the Flexner group; 12 patients were treated with sulfathiazole and 4 with sulfaguanidine.

The authors insist on the following points: early diagnosis and early treatment; high initial dose; adequate subsequent doses divided into fractions every four hours, day and night; usefulness of the ingestion of liquids abundantly; necessity of a strict clinical vigilance and, for further security, hematologic and chemical vigilance; not to persist with this therapy when it is not efficacious during the first days or the first week; not to persist with it over more than two weeks; benefits of vitamin therapy.

The systematic, early and correct treatment of adrenal insufficiency in cases of serious bacillary dysentery is also recommended.

[FROM THE EDITOR'S SUMMARY.]

DISTURBANCES OF NUTRITION, METABOLISM AND THE DIGESTIVE ORGANS IN MALARIA OF THE CHILD. A. ECKSTEIN, Ann. pædiat. **159**:25 (July) 1942.

The following classification of nutritional, metabolic and digestive disturbances in malaria during childhood is proposed:

A. Symptom-poor malaria: (1) in congenital malaria; (2) in acquired malaria.

B. Dystrophy: (1) in congenital malaria; (2) in acquired malaria (*a*) in infancy, (*b*) in early childhood, (*c*) in later childhood (puberty disturbances, dystrophia adiposa genitalis, cachexia).

C. Dyspepsia and enteritis (intestinal form of infant and child malaria): (1) in infancy; (2) in early childhood.

D. Other malarial complications accompanied by diarrhea: (1) toxicosis in infants; (2) cholera form of malaria in older patients; (3) biliary form of malaria; (4) typhoid form of malaria (malaria typhoid). Appendix: typhus abdominalis and malaria.

E. Gastric pains: gastralgia; pseudoappendicitis; pseudoperitonitis.

F. Nephrosis: (1) in malaria quartana; (2) in malaria tropica.

G. Cirrhosis of the liver.

H. Disturbances of the carbohydrate metabolism.

A description is given of the chief clinical symptoms from personal observation (except D 2 and 3). According to the individual age group, characteristic clinical pictures arise, the causal connection of which with malaria may not be generally known. These are always instances of masked malaria, which have a different course than that described for malaria in the textbooks.

Proof is offered that in patients with cirrhosis of the liver the origin is due to malaria tropica. As to the possibility of a disturbance of the carbohydrate metabolsm in attacks of malaria, the fact must be considered that there was an increase of blood sugar in 2 patients with tropical attacks while there was a normal content in 14 patients.

[From the Author's Summary.]

Chronic Infectious Diseases

Third Generation Syphilis: Review of the Recent Literature and Report of a Probable Case. Herman Beerman, Virgene Scherer Wammock and Kathryne Bause Magnuson, Am. J. Syph., Gonor. & Ven. Dis. **26**:504 (July) 1942.

The question of the transmission of syphilis from a parent with congenital syphilis is still debatable. While a moderate number of cases have been reported, very few meet all the criteria necessary for a positive diagnosis. An analysis of the cases of third generation syphilis published in 1933 revealed that in no instance has a really indubitable example of third generation syphilis been reported. The authors state that for a case of third generation syphilis to be authentic the criteria of Fournier and Finger must be fulfilled. These criteria are: 1. Acquired syphilis must be demonstrated in the grandmother and preferably also the grandfather. 2. Prenatal as distinguished from acquired syphilis must be demonstrated in the mother of the infant suspected to have third generation syphilis. Acquired syphilis must be excluded in the case of the mother, and the father must be proved to be healthy. 3. There must be incontestable evidence of prenatal syphilis in the third generation. 4. Manifestations must appear soon after birth in both the second and the third generation.

In this article the authors publish the report of what they believe to be a case of third generation syphilis. The family tree is nicely outlined in a diagram. The grandfather gave a history of a chancre prior to his marriage, in 1905, and was not treated for syphilis until 1920. Serologic reactions of his blood were weakly positive at that time. The grandmother gave a history of receiving forty-seven injections of a "yellow medicine" in her arm in 1921. Her blood gave positive serologic reactions. She had 13 pregnancies. Five of her children, born from 1908 to 1921, died of unknown causes. For 4 of the remaining 8 children a definite diagnosis of congenital syphilis had been made; for 2 there were no data, and 2 had no evidence of syphilis. One of these children was born in 1907 and was first seen in the hospital in 1916 for pneumonia and vaginitis. She was again seen at the age of 12½ years, at which time a diagnosis of congenital syphilis was made. The serologic reactions of her blood were positive. In three years she received fourteen injections of arsenicals and one injection of a mercurial preparation. In 1933 she had an abortion. In 1936 a diagnosis of dementia paralytica was suggested, and examination of the cerebrospinal fluid confirmed this. It continued to do so in spite of large numbers of injections of arsenicals and bismuth and malarial therapy. The patient's husband presented no evidence of syphilis. The results of physical examination and his serologic reactions were negative three times in 1928 and again in 1939. This mother had 6 pregnancies. The first two produced normal children with no evidence of syphilis; the third terminated in an abortion; the fourth produced a child with probable third generation syphilis; the fifth ended in an abortion, and the sixth produced a child with no evidence of syphilis.

In the first 2 pregnancies the mother received treatment. In the fourth pregnancy the mother received no treatment. When this child was 2 months old the reactions of the blood were positive for syphilis. A diagnosis of congenital syphilis was made on the basis of snuffles, lesions on the palms and soles, slight periostitis and positive serologic reactions of the blood. She received adequate antisyphilitic treatment, and her blood repeatedly gave positive serologic reactions until 1941. The cerebrospinal fluid in 1942 gave a negative reaction.

Birdsong, University, Va.

Pleurisy with Effusion. J. L. Bonilla Jr., Am. Rev. Tuberc. **46**:59 (July) 1942.

Among the members of approximately 2,000 families in which at least 1 case of presumed tuberculosis had occurred, 79 instances of pleurisy with effusion were found, excluding patients who were known previously to have had tuberculosis of the reinfection type. Of these 9 were children between the ages of 8 and 11 years and 17 children between 12 to 20 years.

All of the 9 children between the ages of 8 and 11 years had been examined as contacts. Seven had had roentgenograms, and with 1 exception all had been given tuberculin tests. The roentgenogram did not reveal tuberculosis at the time of the first examination of 6 of the 7 patients examined, and the average interval between the roentgenogram and the observation of pleurisy was seven and six-tenths months. In 1 child pulmonary infiltration was observed on the first examination and pleurisy supervened on the same side two months later. For 4 of the 9 children the tuberculin reaction was positive on first examination and pleurisy developed after an average interval of six months. In 1 other case the reaction changed from negative to positive during the period of observation and pleurisy developed three months after the positive reaction was noted.

The outcome of pleurisy as observed in these cases was as follows: One patient was apparently cured after induction of pneumothorax without the development of pulmonary tuberculosis; no treatment was given the remaining 8, and 5 progressed favorably; reinfection type tuberculosis developed in 3, 2 of them dying from this cause.

In both children and adults, the frequency with which filtration occurs, either bilateral or on the side opposite the effusion, especially when it appears after the pleurisy, suggests that in many cases the origin of the infiltration is by way of the blood stream. From the study of those who had tuberculin tests prior to the pleurisy it appears that pleurisy with effusion is a phenomenon without direct relation to primary infection.

EUGENE SMITH, Ogden, Utah.

A COMPARISON OF THE TUBERCULIN PATCH TEST WITH THE MANTOUX TEST. HENRY J. BROCK, New York State J. Med. **42**:1241 (July 1) 1942.

Three hundred and eighty-nine persons were examined by both the patch test and the Mantoux test. Forty-eight persons reacted positively in both tests. One gave a positive reaction to the patch test and a negative reaction to the Mantoux test. (This was the only instance in which there was lack of correlation between the results of the two tests.) The pach test is suitable for children, but slightly positive reactions are more difficult to interpret than in adults.

AIKMAN, Rochester, N. Y.

PREVENTION OF TUBERCULOSIS IN INFANCY. DOROTHY PRICE, Irish J. M. Sc., July 1942, p. 252.

In the author's experience there are two methods of prevention of tuberculosis in infancy: (1) by vaccinating a negative reactor and (2) by prevention of spread from the simple primary complex by early diagnosis. This is done by rest, fresh air, good diet and frequent checks by roentgen examination as to progress.

SNELLING, Toronto, Canada.

FOUR CASES OF PULMONARY TUBERCULOSIS IN ASSOCIATION WITH MENINGITIS. NORAH SCHUSTER, Proc. Roy. Soc. Med. **35**:688 (Aug.) 1942.

In these 4 cases tuberculous meningitis was associated with primary pulmonary lesions, and in none of them was there any sign that the patient had ever had an infection of the alimentary tract. The source of infection was clear in 1 case only; in the others no contact with tuberculosis could be traced. The primary focus was in all cases in or near the apex of the lungs and not in the more characteristic situation, near the interlobar fissures. Meningitis seemed in 2 cases to be a terminal event in hematogenous dissemination and in 2 cases to be due to breakdown of a preexisting focus in the brain as described by Rich and others. A brief history of the 4 cases then follows in the report.

WILLIAMSON, New Orleans.

Diseases of Blood, Heart and Blood Vessels and Spleen

ANOMALOUS CORONARY ARTERIES ARISING FROM THE PULMONARY ARTERY: REPORT OF A CASE IN WHICH THE LEFT CORONARY ARTERY AROSE FROM THE PULMONARY ARTERY. L. A. SOLOFF, Am. Heart J. **24**:118 (July) 1942.

The reported case in which the left coronary artery arose from the pulmonary artery was in an infant of 4½ months. The symptoms were crying after feeding, vomiting, cyanosis and shortness of breath. Autopsy revealed huge aneurysmal dilatation of the left ventricle with degeneration of muscle fibers.

From a review of the literature the author concludes that the origin of both coronary arteries from the pulmonary artery is incompatible with life beyond infancy. If the right coronary artery comes from the pulmonary artery, no symptoms result. If the left coronary artery arises from the pulmonary artery, the condition may be well tolerated, provided there is an adequate collateral circulation. Otherwise, symptoms of angina pectoris and failure of the left ventricle occur.

GIBSON, Chicago.

ISOLATED MYOCARDITIS. O. SAPHIR, Am. Heart J. **24**:167 (Aug.) 1942.

A type of myocarditis of unknown origin, not accompanied by endocarditis or pericarditis, is described. It is found in the absence of demonstrable disease elsewhere in the body. It may occur at any age. Of 13 patients in this series, 3 were children. Death usually occurs suddenly. The diagnosis is rarely made during life.

Dilatation of the heart was found in all instances. Histologic changes were diffuse, chiefly interstitial in location, though the muscle fibers of the heart were also involved. Lymphocytes and endothelial leukocytes were the types of cells most often seen, though polymorphonuclear and eosinophilic leukocytes were also observed.

GIBSON, Chicago.

CHANGES IN COPPER AND IRON RETENTION IN CHRONIC DISEASES ACCOMPANIED BY SECONDARY ANEMIA: II. CHANGES IN LIVER, SPLEEN AND STOMACH. HARRY GROSS, MARTA SANDBERG and OLIVE M. HOLLY, Am. J. M. Sc. **204**:201 (Aug.) 1942.

The authors studied the content of iron and copper in the liver, spleen, stomach and tumor tissue in chronic diseases accompanied by secondary anemia.

There is not only no shortage but actually a large accumulation of iron and copper in the liver and spleen in many cases of anemia. Extreme storage was found in malignant conditions, but it was dependent on some factor other than the anemia.

Insufficient formation of the antianemic factor due to inadequate storage of iron or copper in the stomach, suggested by Sümegi, could not be the factor responsible for the anemia, since at least normal, and in most cases higher than normal, amounts of these elements were present in the stomach.

Tumor tissue showed a high percentage of copper, indicating a strong affinity for this element. This could not, however, have played a role in the production of anemia.

HENSKE, Omaha.

THE PRODUCTION OF CARDIAC LESIONS BY REPEATED INJECTIONS OF DESOXYCORTICOSTERONE ACETATE. DANIEL C. DARROW and HERBERT C. MILLER, J. Clin. Investigation **21**:601 (Sept.) 1942.

Necrosis of the myocardial fibers and replacement by fibroblasts are produced by repeated injections of desoxycorticosterone acetate in rats. The lesions cannot be distinguished from those produced by diets low in potassium. The livers decrease in size after injections of desoxycorticosterone acetate for ten days but are normal in size after four weeks of injections. The cardiac lesions produced by injections of desoxycorticosterone acetate or diets low in potassium can be prevented by addition of potassium chloride to the drinking water. Deficit of body potassium is apparently essential for the production of these lesions.

GUEST, Cincinnati.

THE VELOCITY OF BLOOD FLOW IN INFANTS AND YOUNG CHILDREN, DETERMINED BY RADIOACTIVE SODIUM. JOHN P. HUBBARD, WILLIAM N. PRESTON and RALPH A. ROSS, J. Clin. Investigation **21**:613 (Sept.) 1942.

The velocity of blood flow in young infants and children was measured by determining the time elapsed between the injection of radioactive sodium into a vein of one arm and its arrival in the opposite hand. The latter was signaled by a Geiger counter. For 22 children between 2 and 12 years of age the average circulation time thus determined was eleven seconds, with a range of five to seventeen seconds. For 4 infants between 6 weeks and 22 months of age the average was seven seconds, with a range of three to twelve seconds.

GUEST, Cincinnati.

TRANSPOSITION OF THE GREAT CARDIAC VESSELS. PAUL EMERSON and HYMAN GREEN, J. Pediat. **21**:1 (July) 1942.

The cases of 46 children exhibiting cardiac manifestations with transposition of the great cardiac vessels have been studied clinically and at necropsy. The cases are classified into five principal groups depending on the degree of aortic transposition: (1) overriding aorta with pulmonary stenosis; (2) overriding aorta with pulmonary atresia; (3) simple transposition of the aorta; (4) crossed transposition of the great vessels, and (5) mixed transposition of the great vessels and the tricuspid valve.

The cause is believed to be a malrotation or a counterclockwise rotation of the heart and great vessels; the greater the rotation, the greater is the aortic shift, until in extreme cases the aorta lies over the right ventricle with the tricuspid valve in the left ventricle (group 5).

The difficulty of accurate diagnosis is stressed as many of the patients exhibited symptoms suggesting other than cardiac lesions, namely tense fontanels, stiff and retracted necks, hemorrhage, vomiting and diarrhea. Also difficulty was encountered because of the fact that

few showed a predominating cardiac defect, most having other cardiac anomalies, such as patent ductus arteriosus, septal defect and dextrocardia.

Size of heart, clubbing of fingers and murmurs were not diagnostic. Cyanosis, spells of choking and coughing, dyspnea and failure to gain were common to all. However, certain signs were fairly reliable as an aid to diagnosis. Cyanosis, for example, was most intense and duration of symptoms shortest in simple transposition of the aorta; it was next in order of intensity in cases of overriding aorta with pulmonary atresia. Cyanosis and "spells" came on several months after birth in infants with overriding aorta with pulmonary stenosis, while these signs were present from birth in the other four groups. Anomalies of formation of the head, such as micrognathia, microglossia and choanal atresia, were noticed to be frequent with simple aortic transposition. The blood count was fairly normal in aortic transposition and exhibited pronounced polycythemia in the other groups. Roentgen examination was helpful, particularly in the tetralogy of Fallot (group 1), in which a characteristic sheep's nose deformity is shown, and was of value in groups 4 and 5, in which the aortic arch is shown protruding to the right of the cardiac border.

The ages of patients living the longest in the various groups is of interest. Also given is the average age at death.

	Age of Patients Who Lived Longest	Average Age at Death
Group 1	9 5/12 years	5 months
Group 2	9 2/12 years	7½ months
Group 3	2 1/12 months	5 days
Group 4	5 2/12 years	3½ months
Group 5	4 3/12 years	7½ months

STETTENBENZ, Buffalo.

PROGNOSIS FOR CHILDREN WITH CONGENITAL ANOMALIES OF THE HEART AND CENTRAL VESSELS: LIFE EXPECTANCY IN PATENT DUCTUS ARTERIOSUS. MAY G. WILSON and ROSE LUBSCHEZ, J. Pediat. **21**:23 (July) 1942.

Wilson and Lubschez believe that the risk of surgical ligation of patent ductus arteriosus exceeds the natural risk. They state the hazards of patent ductus arteriosus (uncomplicated) have been overemphasized because of statistics derived mainly from selected fatal cases.

A life table of expectancy was constructed based on a twenty year follow-up of 132 cases of congenital heart disease, the patients ranging in age from 2 to 30 years.

The mortality rate for the first decade is 0.49 per cent; that means that a child in this age group has only 1 chance in 200 of dying before he is 10 years of age. The rate for the second decade is 0.70 per cent; that is, there is 1 chance in 140 of a child's dying before 20 years of age. In the third decade no deaths occurred.

For uncomplicated patent ductus arteriosus the rate was 0.38 per cent; in other words, a child reaching the age of 10 has only 1 chance in 260 of dying before the age of 20.

STETTENBENZ, Buffalo.

CHRONIC MYELOGENOUS LEUCEMIA IN EARLY INFANCY. HENRY G. PONCHER, HOMER F. WEIR and LOUIS R. LIMARZI, J. Pediat. **21**:73 (July) 1942.

A case of chronic myelogenous leukemia in a 6 week old infant is described. The clinical picture (enlarged spleen, anemia and white cell count of 195,000) and myeloid cells seen in blood smears and in material obtained by sternal puncture were typical. Chronic leukemia of early infancy must be differentiated from erythroblastosis fetalis, certain leukemoid reactions of syphilis and other infections and acute leukemia.

STETTENBENZ, Buffalo.

LYMPHOSARCOMATOSIS AND CONGENITAL ABSENCE OF SPLEEN. HERMAN CHARACHE, New York State J. Med. **42**:1363 (July 15) 1942.

Absence of the spleen was found in a man 49 years of age who died of lymphosarcomatosis. There was also complete transposition of all organs of the upper part of the abdomen. No accessory spleen was found. The patient had never been sick and had never been operated on.

AIKMAN, Rochester, N. Y.

A COMPARISON OF AGGLUTININ CONTENT OF RABBIT'S SERUM AFTER INJECTION OF LIQUID AND DRIED SERUM OF RABBITS IMMUNIZED WITH HUMAN GROUP A CELLS. R. JAKOBOWICZ, M. BICH and L. BRYCE, M. J. Australia **2**:143 (Aug. 22) 1942.

The authors found that, judging by the agglutinin titer, liquid serum and dried serum (by hardyization) behave similarly as regards retention in the blood vessels of the recipient.

GONCE, Madison, Wis.

INFECTIOUS MONONUCLEOSIS: A PROBLEM IN DIAGNOSIS. T. E. H. SPARK, M. J. Australia **2**:413 (Nov. 7) 1942.

The author presents a complete discussion of the various aspects of infectious mononucleosis. Case histories of 7 young adults and 3 children (the youngest aged 9 years) are cited.

GONCE JR., Madison, Wis.

THE LEUKEMOID EOSINOPHILIC SYNDROME OF THE INFECTIOUS AFEBRILE TYPE. T. VALLEDOR, F. BALTRONS and L. EXPOSITO, Bol. Soc. cubana de pediat. **14**:18 (Jan.) 1942.

The authors present 5 cases of similar clinical syndromes in children from 2 to 11 years of age. They were clinically characterized by fever of long duration, catarrhal episodes, generalized adenopathies, discrete enlargement of the spleen and intense leukocytosis, with 30,000 to 70,000 white cells. Of the leukocytes 50 to 80 per cent were eosinophils. Most of them were of the adult type. The red cell count was normal in some cases, and a mild form of anemia was found in others. Biopsy of the lymphatic nodes showed follicular hyperplasia and the picture of chronic adenitis. After one to three years all of the patients recovered.

In the differential diagnosis typhoid, Hodgkin's disease, intestinal parasitosis, allergic asthma, dermatosis, hydatid cyst and trichinosis were considered. Findings were negative except for 3 cases in which there were pinworms and Ascaris lumbricoides.

After parasitic treatment the white blood count was unchanged. The authors finally conclude that the eosinophilic syndrome must be considered as a pathologic entity.

SCHLUTZ, Chicago.

DISTURBANCE OF SINUS RHYTHM AND AURICULO-VENTRICULAR CONDUCTION IN A CASE OF TETRALOGY OF FALLOT. R. H. BULLICH and A. PAOLUCCI, Rev. méd. latino-am. **27**:11 (July) 1942.

Electrocardiographic studies on a 17 year old patient with the tetralogy of Fallot, permitted the authors to observe sinus arrhythmia and auriculoventricular conduction, characterized essentially by sinus instability and at certain moments by block. The authors attribute the disturbance in conduction not to organic causes but to anoxemia of the myocardium.

DWYER, Kansas City, Mo.

Diseases of Nose, Throat and Ear

DIAGNOSIS AND MANAGEMENT OF SEVERE INFECTIONS IN INFANTS AND CHILDREN: A REVIEW OF EXPERIENCES SINCE THE INTRODUCTION OF SULFONAMIDE THERAPY: I. SEPSIS OF LATERAL SINUS PHLEBITIS. ALEXIS F. HARTMANN, DOROTHY WOLFF and FRANCES LOVE, J. Pediat. **21**:435 (Oct.) 1942.

Case reports of 19 children suffering from thrombosis of the lateral sinus are presented, including the history of each case, symptoms, physical signs, laboratory data and operative and therapeutic procedures. There were 2 deaths, a mortality of 10.5 per cent, as contrasted with mortality figures of 26 to 35 per cent prior to the days of therapy with sulfonamide compounds. The classic signs of coalescent mastoiditis, complete venous block on one side only and persistent bacteremia, were present in most of the cases but not in all. In this series the results of the Toby-Ayer test were positive in 15 of 19 cases, and in 5 cases the test revealed a sinus block on the side opposite that suspected from the history, observations and roentgenograms. This test is considered to be of great value but is not pathognomonic, since a sinus anomaly may cause a positive reaction and other factors may be present. Organisms were cultured from the blood in 12 of the 19 cases, the hemolytic streptococcus being the predominant organism. It is of interest that in 3 cases neither otologic nor roentgen examination could determine damage to the mastoid process. The great value of the sulfonamide compounds in the preoperative and postoperative management of thrombosis of the lateral sinus is stressed.

STETTENBENZ, Buffalo.

A SIMPLE METHOD FOR TESTING THE HEARING OF SMALL CHILDREN. HARLAN BLOOMER, J. Speech Disorders **7**:311 (Dec.) 1942.

Pure tone audiometer tests are given while pictures of animals and birds are shown, and the child is asked to tell what animal or bird is talking.

PALMER, Wichita, Kan.

RESEARCH CONTRIBUTIONS TO VOICE IMPROVEMENT. WILBERT PRONOVOST, J. Speech Disorders **7**:313 (Dec.) 1942.

Pronovost discusses some of the literature regarding factors influencing quality of voice: expiratory movements, natural pitch, elimination of undue laryngeal tension, inadequate

approximation of the vocal cords, harshness, guttural quality, duration of tones, pitch flexibility, dramatic speech and emphasis. More investigations are needed on voice quality.

PALMER, Wichita, Kan.

EAR PREFERENCE. GORDON E. PETERSON, J. Speech Disorders **7**:319 (Dec.) 1942.

Twenty-four normal children listened to an ear phone set directly in front of the face at a distance of a few inches from, and at the level of, each subject's ears. Three different frequencies were tested on three different dates. When the subject could not hear the sound well, he was told to lean more closely to the ear phone. Normal persons apparently appear to prefer one ear to the other in listening to faint sounds.

PALMER, Wichita, Kan.

THE USE OF SULFADIAZINE IN THE MANAGEMENT OF SIMPLE MASTOIDECTOMY WOUNDS. CHARLES A. TUCKER and CARLYLE G. FLAKE, New England J. Med. **227**:1021 (Dec. 31) 1942.

A controlled series of 41 cases of simple mastoidectomy is presented, divided into three sets as follows: group A, those receiving sulfadiazine locally, the wound being closed without drainage; group B, those receiving sulfadiazine by mouth, the wound being closed without drainage, and group C, those not receiving sulfadiazine, the wound being drained in the usual fashion.

The hospitalization period required for patients in groups A and B averaged around ten days, the wounds healing per primam and the tympanic membrane being healed and dry at that time. The wound broke down in only 1 case in group B. There were no untoward results. The patients in group C had the usual prolonged convalescence.

Those who received the sulfadiazine by mouth did about as well as those who received it locally. The major difference, however, was in healing of the wound, which was definitely improved when the drug was used locally.

GENGENBACH, Denver.

ACUTE OTITIS MEDIA AND MASTOIDITIS. E. M. SEYDELL, South. M. J. **35**:658 (July) 1942.

The author states that when any of the following indications are present operation on the mastoid is usually imperative: (1) formation of a fistula through the skin which may be traced into the mastoid process; (2) formation of a Bezold abscess; (3) subperiosteal abscess over the mastoid or tympanozygomatic region; (4) large amount of infiltration over the mastoid area, with tenderness on pressure; (5) definite drooping of the posterior-superior canal wall; (6) severe hemorrhagic nephritis which does not respond to ordinary treatment and which is associated with a severe otitis media; (7) symptoms of intracranial complications; (8) symptoms of petrositis.

SCHLUTZ, Chicago.

SUBACUTE CATARRHAL OTITIS MEDIA AND MASTOIDITIS WITH EFFUSION. J. M. ROBISON, South. M. J. **35**:815 (Sept.) 1942.

The author offers a classification of catarrhal otitis media and mastoiditis with effusion which regiments these cases clinically according to the presence or absence of coexisting disease.

Some of the physical and chemical characteristics of the effused fluid in the ear in this disease are noted as follows: The effusion in type 1 is an exudate; it has a high protein content (9.95 per cent) with many polymorphonuclear leukocytes. The effusion in type 2 is a transudate. There are a few polymorphonuclear leukocytes; there is a low protein content (1.95), and when the fluid has been present in the tympanum long enough to cause the beginning of the chronic catarrhal stage with its adhesions large mononuclear leukocytes and epithelial cells make their appearance.

The importance of irradiation in the treatment of this disease is stressed.

The tendency of this disease to recur and subside spontaneously, the absence of organisms and purulent changes in the effused fluid and its response to proper treatment lead one to believe that the true cause of this condition is a mechanical one. The basis for such a mechanical theory is as follows: An infection in the sinuses or nasopharynx is followed by inflammation and enlargement of the lateral retropharyngeal lymph nodes and adjacent perinodal tissues. This may result in either a mechanical blockage of the tube by direct pressure or displacement of its axis. The lymph node will swell and act as an obstacle and arrest the normal lymph circulation. The lymphatic trunks above this obstacle dilate; the valves become incompetent, and a retrograde lymph circulation is established. In this manner, lymph channels in the tubal mucosa will dilate, with resulting lymph edema, and produce obstruction of the eustachian tube. This mucosal lymph edema and resulting circulatory change if allowed to persist will eventuate in chronic fibrous stricture of the tubal lumen.

SCHLUTZ, Chicago.

REMARKS ON SOME FUNDAMENTAL PRINCIPLES IN OTO-RHINO-LARYNGOLOGY OF INTEREST IN GENERAL PRACTICE. B. HILLER, M. J. Australia **2**:433 (Nov. 14) 1942.

The natural channel for disposal of nasal secretions being backward into the nasopharynx, the negative pressure obtained by "hawking" is the normal, even if not esthetic, method of removing secretions from the nose. Forcible blowing of the nose in the presence of infection of the upper respiratory tract is a common cause of otitis media and sinusitis. Because the eustachian tube of the infant is shorter, of larger lumen and more horizontally placed than that of the adult, his being in the dorsal recumbent position while feeding is a common cause of acute suppuration of the middle ear. The local use of ephedrine, epinephrine and other similar drugs for the relief of intranasal congestion is contrary to the principle of increase in circulation applied to any acute inflammatory condition elsewhere in the body. Inhalations, by increasing the usual rhinorrhea, stimulate and assist nature in the normal way to clear up the condition, whereas the vasoconstrictors decrease the discharge and the circulation to the part, and so hinder the natural processes of recovery. GONCE JR., Madison, Wis.

Diseases of Lungs, Pleura and Mediastinum

BRONCHIAL OBSTRUCTION AND TRACHEOBRONCHIAL TUBERCULOSIS. EDWARD B. BENEDICT. New England J. Med. **227**:1014 (Dec. 31) 1942.

The author presents a study of 3 case histories, with the following conclusions: Among the most frequent causes of bronchial obstruction are carcinoma, benign tumor and tuberculosis. The clinical picture may lead to an erroneous presumptive diagnosis, for the underlying cause of the obstruction frequently cannot be determined by laboratory and roentgen studies. Bronchoscopy with biopsy or smear is necessary to establish a positive diagnosis.

GENGENBACH, Denver.

THE MEDICAL AND SURGICAL MANAGEMENT OF EMPYEMA IN CHILDHOOD. SAMUEL L. ELLENBERG, New York State J. Med. **42**:1243 (July 1) 1942.

Ellenberg reports 115 cases of empyema in children under 15 years of age. In 14 cases aspiration of the chest alone was used, and all of the patients recovered. There were 29 cases in which intercostal drainage was instituted, with three deaths, and 62 cases in which treatment was by means of rib resection, the mortality not being stated.

In the cases in which sulfapyridine was used, better results were obtained. Transfusions were given to 50 patients. Ellenberg advocates the conservative plan of therapy. The incidence of empyema was found definitely higher in the Negro race than in the white.

AIKMAN, Rochester, N. Y.

CANCER OF THE LUNG IN INFANCY. HARRY HAUSER, Radiology **39**:33 (July) 1942.

The case of a Negro girl 17 months of age with a primary cancer of the left lung is reported. For five months there had been a dry cough, thought to be "whooping cough." The symptoms cleared but recurred later, with the cough becoming worse. There was no hemoptysis. The weight remained stationary. The left lung was dull to percussion.

Roentgen examination of the chest (anteroposterior projection) showed the entire left hemithorax to be the site of increased density of a homogeneous character. The heart and trachea were considerably displaced to the right. The roentgen findings were attributed to a large amount of fluid in the left pleural cavity.

Death occurred apparently six months after the first symptoms. At necropsy there was no fluid in the pleural cavities. A tumor of the left lung accounted for the dense shadow on the roentgenogram and displacement of the heart to the right. The pathologic diagnosis was (1) small cell carcinoma; (2) bronchopneumonia.

The author emphasizes the rarity of primary cancer in infancy. ANSPACH, Chicago.

SIGHING DYSPNOEA AND THE HYPERVENTILATION SYNDROME. M. D. SILBERBERG, M. J. Australia **2**:38 (July 18) 1942.

In Silberberg's experience, sighing dyspnea is a common form of functional breathlessness which is frequently mistaken for the breathlessness of cardiac, pulmonary or thyroid disease. It is more common in females than in males; it occurs in children and adolescents but is more usual in adults up to the fifth decade, especially in obese, emphysematous middle-aged patients whose respiratory muscles function badly. In sighing dyspnea, inquiry elicits from

the patient that there is a sensation of unsatisfied breathing. This type of breathlessness is related, not to exertion, but to fatigue and emotion. Occasionally it is excessive and produces the hyperventilation syndrome, which may culminate in tetany. GONCE, Madison, Wis.

THE PHYSICAL SIGNS OF A PLEURAL COLLECTION, WITH PARTICULAR REGARD TO A NEW SIGN FOR ITS DETECTION. M. BERAH, M. J. Australia **2**:463 (Nov. 21) 1942.

In the presence of fluid in the pleural cavity, percussion on the spine with the patient sitting up in bed reveals an area of dulness the height of which is proportionate to the amount of fluid. If the patient is now asked to lie down on his sound side, one would expect an increase of dulness in the spine; but the opposite happens, as the area of dulness is reduced by 1, 2 or 3 inches (2.5, 5 or 7.5 cm.). This paradox, described and explained by D'Amato, appears because the fluid, instead of flowing backward, fills the pleuropericardial sinus.

GONCE JR., Madison, Wis.

THE ASPHYXIAL SYNDROME. ROBERTO MACHEDO and FRANCISCO BORGES, Arch. de med. inf. **11**:131 (July-Sept.) 1942.

This syndrome of dyspnea, stridor, perspiration and grayish skin was observed in a child of 28 days. The patient recovered after a tracheotomy. The only cause found was hypertrophy of the right lobe of the thymus. SANFORD, Chicago.

THE CLINICAL PICTURE OF INTERSTITIAL PLASMA CELL PNEUMONIA OF THE INFANT. ERNST HUG, Ann. pædiat. **159**:2 (July) 1942.

Hug presents a clinical description of 8 cases of plasma cell interstitial pneumonia in infants. All of the patients were born prematurely. The results of the physical, roentgen and other clinical methods of examination are given and compared with the results from publications concerning this disease of the lungs.

Concerning the hitherto unexplained question of causation, the opinion is expressed that interstitial plasma cell pneumonia is due to bad technics of feeding; infection is not considered probable. The damage arises through repeated small aspirations of food in consequence of careless feeding technic of prematurely born children, difficult to feed. This opinion is confirmed by the postmortem status found by Roulet. The sex relation of the illness results from its nature as an injury due to lack of care.

Short wave treatment has proved efficacious to a certain degree, while the administration of sulfathiazole has failed. [FROM THE AUTHOR'S SUMMARY.]

Diseases of the Gastrointestinal Tract, Liver and Peritoneum

THE EFFECT OF SULFANILAMIDE ON THE EXPERIMENTALLY DAMAGED LIVER. THOMAS E. MACHELLA and GEORGE M. HIGGINS, Am. J. M. Sc. **204**:194 (Aug.) 1942.

The authors demonstrated that the administration of sulfanilamide to a group of rats with hepatitis produced by inhalation of carbon tetrachloride did not increase the damage to the liver. On the contrary, it appeared to lessen the damage.

After induction of hepatitis by means of carbon tetrachloride, sulfanilamide did not impede regeneration of the liver after the administration of carbon tetrachloride had been discontinued.

The simultaneous administration of alcohol and sulfanilamide to rats did not result in damage of the hepatic cells, although in some of the animals that received only the alcohol, fat was deposited in the liver.

The administration of sulfanilamide to animals that had obstructive jaundice did not produce any detectable injury to the liver not ascribable to biliary obstruction.

HENSKE, Omaha.

NUTRITION: THE APPEARANCE OF THE TONGUE AS AN INDEX OF NUTRITIONAL DEFICIENCY. HAROLD JEGHERS, New England J. Med. **227**:221 (Aug. 6) 1942.

This is a lengthy article explaining in detail the diagnostic importance of the appearance of the tongue. Some of the subjects treated are: dry and coated tongue; pellagrous and riboflavin deficiency glossitis; glossitis from other deficiencies; Hunter's, Moeller's and atrophic glossitis; wandering rash of the tongue, and grooved and black, hairy tongue.

GENGENBACH, Denver.

AEROESOPHOGRAPHY IN CONGENITAL ATRESIA OF THE ESOPHAGUS. MURRAY FUHRMAN, DAVID DRAGUTSKY, GEORGE ROOK and LEO GROSSMAN, Radiology **39**:100 (July) 1942.

The authors publish roentgenograms of a 36 hour old infant which show a ballooned-out esophagus above the atresia demonstrated by means of injection of air through a soft rubber catheter introduced into the upper portion of the esophagus. Since in this type of congenital atresia barium administered orally is most likely to spill over into the trachea and bronchial tree, the authors have used air instead of barium. This procedure was suggested by them in a previous report.

ANSPACH, Chicago.

MEDICAL OR SURGICAL TREATMENT OF PYLORIC STENOSIS. STEFAN ENGEL, Brit. J. Child. Dis. **39**:72 (July-Sept.) 1942.

Engel considers the muscle less of a factor in the production of pyloric stenosis than the mucous membrane. In the relaxed pylorus the folds of mucous membrane decrease, whereas in the contracted muscle they increase. Pylorotomy diminishes the folds by letting a portion of the mucous membrane escape from the narrow pyloric channel.

The author divides the cases of pyloric stenosis into four grades: (1) slight; (2) medium; (3) severe, and (4) very severe.

He believes that grades 1 and 2 may be treated medically and also grade 3 under close supervision but that grade 4 requires an operation as soon as possible.

Atropine and atropine methylnitrate, usually supposed to affect the pyloric muscle, may also influence the mucous membrane. Diet, local treatment of the stomach and in particular good nursing help to tide over the period of stenosis. If the degree of stenosis is slight, atropine methylnitrate is not necessary.

LANGMANN, New York.

HIRSCHSPRUNG'S DISEASE TREATED BY SPINAL ANÆSTHESIA. DONALD COURT and J. K. HALSER, Proc. Roy. Soc. Med. **35**:687 (Aug.) 1942.

The patient, a boy of 8 years, had been constipated since birth. In the first two years he would go four or five weeks without defecation. He had been given liquid petrolatum regularly and intermittent enemas, which had produced small motions but had had little effect on the abdominal enlargement. On examination the abdomen was distended and tympanitic and showed visible coils of large intestine. The circumference around the umbilicus was 30 inches (76 cm.). He was given sufficient spinal anesthetic to paralyze the anterior nerve roots up to and including the sixth dorsal segment. He was given two treatments ten days apart. Two days after the last, normal bowel movement began and continued until his discharge from the hospital two weeks later. Seven months later the circumference of his abdomen was 23½ inches (60 cm.) and there were one or two large bowel movements a day. The details of the treatment are given in the report.

WILLIAMSON, New Orleans.

Nervous Diseases

LISSENCEPHALY. A. EARL WALKER, Arch. Neurol. & Psychiat. **48**:13 (July) 1942.

Lissencephaly is a rare condition of the brain in which development stops at an early stage (about 3 months). Walker reports a case of this disorder (5 cases have been previously reported). There were hydrocephalus, smooth cerebral hemispheres (agyria), absence of the corpus callosum, poorly developed subarachnoid space and maldevelopment of the cerebrum, cerebellum and both eyes.

BEVERLY, Chicago.

THE ARNOLD-CHIARI MALFORMATION. M. A. OGRYZLO, Arch. Neurol. & Psychiat. **48**:30 (July) 1942.

This is another report on the Arnold-Chiari malformation. This is a deformity of the hindbrain in which there is displacement of parts of the cerebellum and brain stem through the foramen magnum. The author reports 7 cases, 4 in infants and 3 in adults. The adults were operated on, with fatality in 1 case.

BEVERLY, Chicago.

PROGRESSIVE DEGENERATIVE ENCEPHALOPATHY: OCCURRENCE IN INFANCY, WITH ANTENATAL ONSET SIMULATING "SWAYBACK" OF LAMBS; REPORT OF A CASE. N. W. WINKELMAN and MATTHEW T. MOORE, Arch. Neurol. & Psychiat. **48**:54 (July) 1942.

"1. In an infant born partially asphyxiated after a prolonged labor there developed progressive spastic diplegia, occasional convulsive seizures and marasmus. Death occurred within twelve weeks of pneumonia and purulent meningitis. The brain showed maldevelopment,

subcortical demyelination, liquefaction necrosis with cyst formation and underdevelopment and degeneration of the cortex.

"2. The clinical and pathologic aspects of the condition in this case resemble in most details those of 'swayback' in lambs.

"3. In view of the fact that 'swayback' has been shown to develop in utero and that in our case comparable signs and symptoms were apparent at the time of birth, the conclusion is justified that in our case the condition was of antenatal origin.

"4. The features in this case may help to strengthen further the viewpoint of those who believe that antenatal factors may explain some of the clinical syndromes often attributed to birth trauma."

AUTHORS' SUMMARY.

ENCEPHALOMALACIA WITH CAVITY FORMATION IN INFANTS. LEWIS D. STEVENSON and LILLIAN E. MCGOWAN, Arch. Path. **34**:286 (July) 1942.

Stevenson and McGowan describe their observations on 7 infants with what they term "encephalomalacia with cavity formation," mainly within the white matter. The cause is unknown; in 3 of their cases the Wassermann reaction was positive. The condition does not depend on thrombosis of venous sinuses, on an arterial abnormality of the brain or on encephalitis.

It was present at birth, and the clinical picture developed shortly thereafter. There is no characteristic clinical syndrome, but the condition can be diagnosed during the life of the infant by encephalogram, which shows enlargement of the ventricles and in many cases filling of the subcortical cavities with air.

WINKELMAN, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

ANOTHER CASE OF TORULA MENINGITIS. B. F. SAMPSON and J. E. FARREN, South African M. J. **16**:245 (July 11) 1942.

The authors report a case of torula meningitis in an Indian youth aged 14 years in which for five months the only symptom was headache. Administration of sulfapyridine resulted in a slight improvement, which was not maintained. Death occurred in six months from the onset of the illness. The case is interesting in view of the typical early history of refractory headache of gradual onset and of the diagnosis being arrived at through seeing the phenomenon of a budding "lymphocyte." It is pointed out that if the torula organisms are taken to be lymphocytes a diagnosis of benign lymphocytic meningitis may easily be made, especially in the early weeks of the illness when the clinical picture looks benign.

GONCE, Madison, Wis.

MENINGITIS DUE TO HAEMOPHILUS INFLUENZAE IN EARLY INFANCY. D. F. SOSA BENS, Bol. Soc. cubana de pediat. **14**:331 (July) 1942.

The author reports on 9 cases of this type of meningitis. All the infants were under 22 months old.

The therapy employed consisted in the use of sulfapyridine or sulfathiazole orally and intravenous injection of the sodium salts of these two preparations in 2 of the cases; in 1 case an anti-influenza serum (type B) was used in addition to the drugs.

He points out that this form of meningitis is frequent in hospitalized patients of this age range, that most of them have very little natural immunity and that nutritional factors, on account of poor environment, play an important role.

One patient recovered completely (this was an infant aged 13 months who received large doses of sulfathiazole [213 Gm. in forty-three days]). In 1 of the patients the author was definitely under the impression that the sulfathiazole had a toxic effect and probably contributed to a fatal termination.

The use of sulfapyridine alone in doses of 0.37 to 0.44 Gm. per pound (0.5 Kg.) of body weight in twenty-four hours seemed in no way to arrest the disease or cause any improvement. A combination with sulfathiazole brought about the recovery of 1 infant and in the case of another there was some improvement and some delay in the fatal termination of the process. The tolerance to sulfanilamide seems to be elevated at this age.

The highest concentration observed was 11 mg. per hundred cubic centimeters of blood. The author did not observe any unfavorable effects on the blood picture in the series of cases reported. He points out, however, that all of the patients received repeated blood transfusions. Oliguria, tremors and discrete cutaneous eruptions were observed.

The author believes that the mixed treatment, combining the use of specific serum and a sulfonamide compound, offers the best promise in the treatment of this condition, although the results are not striking in early infancy.

SCHLUTZ, Chicago.

PURULENT MENINGITIS DUE TO SALMONELLA EASTBOURNE IN AN INFANT OF THREE MONTHS. T. VALLEDOR, E. G. PEREZ and J. PEDRERA, Bol. Soc. cubana de pediat. **14**:413 (Aug.) 1942.

The author reports this rare type of infection in an infant aged 3 months. The patient was treated with large doses of sulfadiazine, repeated blood transfusions and vitamin therapy. The severe symptoms regressed rapidly, and an apparent cure was effected within sixty-two days.

A remission occurred after the first week of treatment but was promptly controlled with sulfadiazine. The infant seemed to show some residual symptoms suggesting the possibility that there might be a chronic encephalitic process; there were some motor disturbances, and there was a slight retardation in growth.

The sulfadiazine seemed to be well tolerated in spite of the large doses used. It did not produce anemia or other drug effects. The child showed pronounced eosinophilia.

SCHLUTZ, Chicago.

Psychology and Psychiatry

PNEUMOENCEPHALOGRAPHY AND THE DEVELOPMENTAL DIAGNOSIS OF BEHAVIOR. CATHERINE S. AMATRUDA, J. Pediat. **21**:147 (Aug.) 1942.

The author collected 53 cases in which it was possible to correlate the results of developmental examinations with those of pneumoencephalographic examinations. On the basis of the conditions noted on clinical examination the cases were presented in the following groups: (A) congenital malformations and aplasias; (B) cerebral degenerative disease; (C) epilepsy; (D) hydrocephalus; (E) cerebral injury; (F) postmeningitis; (G) postencephalitis. A correlation of cerebral damage revealed by roentgenograms and developmental status and behavior was then attempted.

Group A.—This comprised cases of various malformations, such as porencephaly, hypertelorism, synostosis and agenesis of the corpus callosum. All were characterized by lack of normal progress and development. Roentgenograms showed atrophy of the cortex and ventricular dilatation.

Group B.—In this group were cases in which normal birth and development were followed by convulsions, degeneration and subsequent idiocy, usually evident at from 3 to 6 months of age. Roentgenograms showed severe atrophy of the cortex and ventricular dilatation. Prognosis for all was poor.

Group C.—The cases of epilepsy were the most interesting of all, since in most instances atrophy of the cortex was revealed but developmental behavior was usually normal (in marked contrast to groups A and B). In other words, cortical atrophy does not necessarily mean mental deterioration or a bad prognosis. Probably in groups A and B definite failure of cerebral growth occurred, whereas in group C normal growth occurred but the brain was compressed by increased intracranial pressure.

Group D.—Here again in certain types of communicating hydrocephalus atrophy of the cortex may not cause failure of normal behavior development.

Group E.—Cases of cerebral injury include those involving serious difficulty at birth and during the neonatal period with marked neuromotor changes. Roentgenograms show atrophy, ventricular dilatation and even porencephaly, confirming the diagnosis. In these cases the outcome is uncertain, but possible normal mental development may occur in face of obvious damage to the brain.

Group F.—The results were inconclusive, but again severe cortical atrophy from postmeningitis hydrocephalus may not cause mental damage.

Group G.—Only 5 cases of encephalitis are reported, and the results are indeterminate.

Conclusion.—The author states: "In the presence of normal behavior, pneumoencephalographic evidence of cortical atrophy, so-called, is prognostically meaningless because behavior is the final criterion of the functional integrity of the nervous system."

STETTENBENZ, Buffalo.

THE FREQUENCY OF STUTTERING IN RELATION TO WORD LENGTH DURING ORAL READING. SPENCER F. BROWN and ADELAIDE MOREN, J. Speech Disorders **7**:153 (Aug.) 1942.

Thirty-two stutterers read 10,000 words of simple factual material to a statistically reliable experimenter, who recorded spasms of stuttering and other speech errors on a second copy of the material. The following conclusions may be drawn from the data: 1. Longer adjectives and prepositions are more difficult for stutterers than are shorter ones, whether syllable-letter,

syllable or letter categories are used as a basis for measuring length of the word. 2. The factor of phonetic difficulty partially explains this, but not all variations can be so explained. 3. All other factors being equal, the amount of stuttering tends to vary directly with word length. The authors suggest that the reason for this phenomenon may be the greater prominence in speech of longer words and the stutterer's consequent desire to avoid stuttering at points where these words occur.

PALMER, Wichita, Kan.

AN EXPERIMENTAL STUDY IN MEASURING AND MODIFYING ASSERTIVE BEHAVIOR IN YOUNG CHILDREN. GERTRUDE E. CHITTENDEN, Monograph of the Society for Research in Child Development, National Research Council, 1942, vol. 7, series 31, no. 1.

Two types of assertive behavior, domination and cooperation, were observed in 88 preschool children during five test situations of five minutes each in which only one toy was made available to 2 children. Each child was paired with 5 different companions.

A measure of internal consistency applied to the five partial scores in the two types of assertiveness indicated a higher intercorrelation for domination than for cooperation. From this observation the investigator formulated the hypothesis that the dominative child makes less adaptation of his behavior to the change in companion than does the cooperative child.

With this hypothesis as a basis, a training program was set up to help the dominative child understand social situations and adapt his behavior in light of his understanding. The training program, in which 10 highly dominative children participated, consisted of a series of "plays," during which dolls, playing the roles of preschool children, participated in situations and solved social problems similar to those experienced by children in the nursery school. A control group of 9 highly dominative children were not trained.

After the training period each trained and each control child was retested in the initial test situation. A comparison of these scores by the method of analysis of covariance showed that the trained children were significantly less dominative than the controls. The increase in cooperation for the trained children was not statistically significant. A comparison of scores obtained from observation of the trained children in the preschool free play situation showed them to be significantly less dominative and significantly more cooperative after training.

[AUTHOR'S SUMMARY]

PRACTICAL PSYCHIATRY WITH ADOLESCENTS. DANIEL J. SULLIVAN and OTTO BILLIG, New England J. Med. **227**:253 (Aug. 13) 1942.

Explanation, reassurance and desensitization are direct methods of psychotherapy, but the indirect methods, such as a daily schedule of activities to fill up the adolescent's day, a modification of parental attitudes and major or minor changes in school environment, are often as helpful. Hobbies should be of indoor and outdoor and of individual and group types.

Group activities should occupy a large part of the adolescent's time, since it is vital for him to learn how to get along with others. The patient's keeping of a diary is useful in selected cases to enable the investigator to get a better understanding of the adolescent, who can often write about his troubles better than he can talk of them.

Modifying the family situation is necessary in practically every case, this procedure being omitted when the adolescent comes spontaneously and sometimes secretly so far as the family are concerned. The fault is seldom all on the adolescent's side; often, all he needs is a little wise neglect and absence of parental overattention.

A disconcerting fact, but one that does not negate the recommendations just given, is the observation that the parents are essential in preventing the development of criminally delinquent adolescents. The Gluecks found that 60 per cent of 500 young criminals and 58 per cent of 500 delinquent women had a background of a broken home. "It may frequently be the physician who can best serve as parent-substitute, and particularly the pediatrician, if he has dealt wisely with his patients during their childhood. In this terra nova of medical interest he has early staked his claim."

Sex education should have been taken care of by the parents during the childhood years, but it is a wise rule to get from the adolescent his ideas on anatomy, physiology, coitus, pregnancy, childbirth and venereal disease. The most obvious obstacle during adolescence is the acquisition of heterosexual stability. Despite the so-called "frankness" of modern youth, the adolescent is often reluctant to admit that he is troubled about sexual matters. Aldrich and Aldrich wrote as follows: "An appreciation of sexual competency as a constructive asset is a high and difficult standard for children to attain in our inconsistent world."

Career ambition needs attention, not just actually to pick out the one best suited for the particular adolescent but also to keep him interested in the future along with a reasonable amount of self evaluation.

Delinquency refers to antisocial behavior that is considered criminal, such as habitual stealing, destruction of property, vagabondage and running away, occurring in children under the age of 16 years in most states.

Feelings of inferiority may be inferred from the adolescent's behavior (aggressiveness, noisy conduct, shyness and aloofness), or the cause may be described by him as frail body, clumsiness, stupidity, inability to dance, acne, inadequate clothes and so forth. Consultation with the school authorities may be advisable when behavior or scholastic difficulties beset the patient. Personality clashes between teacher and adolescent should not be taken seriously provided investigation does not show the teacher to be a misfit. The adolescent must be quietly impressed with the reality that all through life he will be under the authority of apparently disagreeable persons and must learn to "take it."

Marriage of an adolescent does not automatically make him an adult, no matter what the legal attitude may be. The adolescents are grateful for advice concerning special adjustment. In turn, the parents need desensitization to their "in-laws."

Dishonesty, including lying, cheating in examinations and stealing, is important, since etiologic treatment is always best.

Concerning psychotherapy in general, the following realistic evaluation of Kanner is pertinent: "Practical psychotherapy is the organized treatment of a person in trouble, uniquely adapted and readapted to a unique person and a unique situation. . . . It steers clear both of Pollyanna sweetness and nihilistic bitterness. . . ."

GENGENBACH, Denver.

CORRECTIVE ASPECTS OF STAMMERING. T. G. LEARY, M. J. Australia **2**:119 (Aug. 15) 1942.

Stammering is not a disorder of the voice or of breathing; it is a disorder of incoordination of speech centers of the brain. Recurring failures produce an expectation to stammer, with resultant strain and loss of confidence. The stammerer is imbued with the idea that he cannot speak. It is in essence a fear complex that has developed from a succession of humiliating failures. People do not stammer because they are nervous; the stammer is the cause of the nervousness. Corrective measures aim at the readaptation of the stammerer to his environment by the recreation of his personality. The patient must, in fact, make a fresh beginning. Stammerers commencing treatment can sometimes hardly speak at all, owing to the conditions of strain. They are obviously in need of relaxation and require the simplest and most elemental way of speaking. When they are sufficiently relaxed, they are induced to speak in a rhythmic cadence, with lengthened and full vowel sounds. It will be found necessary, to maintain a smooth flow of rhythm, that arms and legs be used to assist in keeping time. Far reaching and beneficial results will be obtained from the rhythm in stilling the psychic conflict and rendering the personality expressive. The patient will lose his inferiority complex and his repression. The subsequent history of the patient will have relation to his strength of purpose and to his happiness in his environment.

GONCE, Madison, Wis.

Diseases of the Genitourinary Tract

THE EFFECT OF KIDNEY POSITION ON RENAL BLOOD FLOW AND FUNCTION. D. J. GABRIELE, Am. J. M. Sc. **204**:227 (Aug.) 1942.

The author shows that renal blood flow is profoundly reduced when the piezometric angle (the angle between a tributary and the advancing blood stream in the mother artery) of the renal artery is made relatively acute.

It is possible that the anatomic basis of predisposition toward hypertension lies in the extreme variability of the position of the kidney.

The deranged renal function of uncomplicated late toxemia of pregnancy may be the result of relatively high elevation of the kidneys during gestation, depending on the body type and the tone of the abdominal muscles.

HENSKE, Omaha.

THE SERUM ANTISTREPTOLYSIN TITER IN CHRONIC GLOMERULONEPRITIS. DAVID P. EARLE JR., EMILY N. LOEB, DAVID SEEGAL, JOHN D. LYTTLE and ELIZABETH L. JOST, J. Clin. Investigation **21**:483 (July) 1942.

In a series of 81 patients with chronic glomerulonephritis in which the antistreptolysin titer response of the serum was studied over periods of from four months to eight years, it was found that the greater the magnitude of the rise in antistreptolysin titer, the greater was the frequency of positive cultures for group A hemolytic streptococci. The incidence of preceding acute infections was higher in the group showing larger rises in titer than among those exhibiting the medium and small rises.

Certain factors affecting the antistreptolysin titer response in chronic glomerulonephritis were examined, with the following results: (a) Sex had no effect; (b) children generally exhibited greater titer response to infection than did adults; (c) the character of the preceding hemolytic streptococcus infection did not appear to affect the magnitude of the titer response; (d) patients with "nephrotic" edema generally exhibited smaller rises in titer than did those without edema.

GUEST, Cincinnati.

THE RELATION OF THE SERUM ANTISTREPTOLYSIN TITER TO THE EXACERBATION IN CHRONIC GLOMERULONEPHRITIS. DAVID P. EARLE JR., DAVID SEEGAL, JOHN D. LYTTLE, EMILY N. LOEB and ELIZABETH L. JOST, J. Clin. Investigation **21**:491 (July) 1942.

In 81 nephritic patients, studied for from four months to eight years, there were observed 33 exacerbations of chronic glomerulonephritis. Each exacerbation was preceded by an infection. Twenty-four of the exacerbations were associated with rises in serum antistreptolysin titer. The exacerbation preceded the onset of the rise in antistreptolysin titer in 7 of the 8 sufficiently studied instances.

GUEST, Cincinnati.

THE UREA CLEARANCE OF YOUNG PREMATURE AND FULL TERM INFANTS. HARRY H. GORDON, HAROLD E. HARRISON and HELEN MCNAMARA, J. Clin. Investigation **21**:499 (July) 1942.

The twenty-four hour plasma clearance of urea was determined in 35 observations on 15 premature infants and in 12 observations on 9 full term infants, ranging in age from 8 to 65 and from 7 to 73 days respectively. The urea clearance is lower in premature than in full term infants, and in both groups of young infants it is lower than that reported for older subjects. No relation was found to exist between urine flow and urea clearance.

[AUTHORS' SUMMARY.]

EXPERIMENTAL HUMAN VAGINAL TRICHOMONIASIS. H. CLOSE HESSELTINE, S. L. WOLTERS and ALICE CAMPBELL, J. Infect. Dis. **71**:127 (Sept.-Oct.) 1942.

"These observations confirm those of Trussell and Plass to the extent that the clinical entity of vaginal trichomoniasis can be produced by the inoculation of the human vagina with the Trussell-Plass culture of vaginal trichomonads." The modus operandi is unknown.

The bacterial flora and cell forms in the vagina seem related in some way to the disease entity; therefore, it may be assumed that some women are more likely to acquire the infection than others when similarly exposed.

These observations suggest that the more resistant or persistent infections have some connection with the physiologic state of the vagina and are consistent with the present day therapy, namely restoration of the normal physiologic state of the vagina and prevention of reinfection while the patient is under treatment.

TOOMEY, Cleveland.

NEPHROTIC DWARFISM WITH GLYCOSURIA AND RICKETS OF THE LOW PHOSPHORUS TYPE. R. AIDIN and E. NOBEL, Brit. J. Child. Dis. **39**:65 (July-Sept.) 1942.

The characteristics of Fanconi's syndrome are early dwarfism beginning in infancy, rickets, thermolability, proteinuria, glycosuria with no fasting hyperglycemia and hypophosphatemia. There is normal or slightly increased content of nitrogen in the blood, and acetone is absent from the urine.

The case of an 8 year old girl is reported. Vitamin D had no effect on the rachitic condition of this patient's bones. The condition of this child differed from Fanconi's syndrome in that there was no glycolability, the fasting blood sugar was never under 100 mg. per hundred cubic centimeters and the test for tolerance of dextrose showed a diabetic plateau.

LANGMANN, New York.

NEPHROPATHIES OF THE CHILD: A STUDY OF THE URINARY SEDIMENT BY THE METHOD OF ADDIS. E. GALÁN CONESA, Bol. Soc. cubana de pediat. **14**:387 (Aug.) 1942.

The author reports on the usefulness of the Addis sedimentation count for diagnosis of nephritis and bases his study on a three year observation of 31 children affected with this disorder. The cases are classified according to clinical picture, the chemistry of the blood plasma and the sedimentation rate.

He points out that the Addis count is not necessary for the diagnosis of typical hemorrhagic nephritis, for a classic nephrotic syndrome or for a well established terminal stage of chronic nephritis. On the other hand, its use is very important for: (*a*) the diagnosis of the subclinical types of nephritis and (*b*) the diagnosis of a latent stage of an undiscovered nephritis. This is the most valuable information to be obtained from the work of Addis.

In conclusion, the author states that when a routine urinalysis shows some abnormality or if there is any clinical suspicion of nephritis the sediment of the urine should be studied by the means of the Addis count. It is in his opinion the best method for arriving at a diagnosis or for discovering clinical facts that usually escape the average clinical observer or that fail to show up with other laboratory methods of diagnosis.

SCHLUTZ, Chicago.

Diseases of the Ductless Glands; Endocrinology

THE INHERITANCE OF DIABETES INSIPIDUS. HARRY BLOTNER, Am. J. M. Sc. **204**:261 (Aug.) 1942.

The author reviews the literature on familial diabetes insipidus and presents the cases of a boy, his mother and his maternal grandmother, all of whom had diabetes insipidus.

From case records, he concludes that diabetes insipidus is a disease that may be inherited and transmitted through many generations of a family through the maternal or the paternal side to either male or female children.

HENSKE, Omaha.

INFLUENCE OF ADRENALECTOMY UPON THE RATE OF GLUCOSE ABSORPTION FROM THE INTESTINE. WILLIAM G. CLARK and EATON M. MACKAY, Am. J. Physiol. **137**:104 (Aug.) 1942.

There has been disagreement as to whether or not adrenalectomy has a specific effect on the absorption of dextrose from the intestinal tract. The authors observed the influence of adrenalectomy on the absorption rate for dextrose in intact, unanesthetized animals. They conclude that the impaired ability to absorb dextrose evident in adrenalectomized animals under some conditions is not referable to the lack of the influence of any adrenal cortex hormone on the intestinal mucosa but rather to the consequent disturbance of sodium chloride metabolism.

NOURSE, Cleveland.

EFFECT OF EQUINE GONADOTROPIN ON TESTES OF HYPOPHYSECTOMIZED MONKEYS. PHILIP E. SMITH, Endocrinology **31**:1 (July) 1942.

Experiments on hypophysectomized monkeys indicate that the maintenance and restorative effects of gonadotropin from the serum of pregnant mares are disappointing and are in contrast to the pronounced gonadotropic effects reported in hypophysectomized rats. The maintenance and restorative effects in monkeys are, in fact, not equal to those given by large dosages of androgen. The shortness of the period during which the gonadotropin was effective appears to be due to its antigenicity.

JACOBSEN, Buffalo.

EFFECT OF EARLY HYPOPHYSECTOMY ON HYPOTHALAMIC OBESITY. A. W. HETHERINGTON and S. W. RANSON, Endocrinology **31**:30 (July) 1942.

In rats obesity can be produced by fairly large symmetric lesions which destroy the region of the ventromedial hypothalamic nuclei bilaterally or interrupt at successively more caudal levels a large number of the descending fibers from the hypothalamic cell groups. Taken together with the evidence that hypophysectomy, stalk section or destruction of the infundibular region does not cause adiposity, the results here reported are interpreted as indicating that hypothalamic control of fat metabolism and the dysfunction of that control which leads to adiposity do not depend on the hypophysis.

JACOBSEN, Buffalo.

GROWTH IN SILVER DWARF MICE, WITH AND WITHOUT INJECTIONS OF ANTERIOR PITUITARY EXTRACTS. ROBERT W. BATES, THEOPHIL LAANWS, E. C. MACDOWELL and OSCAR RIDDLE, Endocrinology **31**:53 (July) 1942.

Injection for thirty days of as little as 1 microgram daily of two recombined anterior pituitary fractions—one a good prolactin and the other practically free of prolactin—caused a 30 per cent gain in the body weight of silver dwarf mice.

JACOBSEN, Buffalo.

EFFECT OF RAT'S LIVER ON ACTIVITY OF TESTOSTERONE AND METHYL TESTOSTERONE. M. W. BURRILL and R. R. GREENE, Endocrinology **31**:73 (July) 1942.

The ability of the rat's liver to inactivate testosterone and methyl testosterone was tested by implanting into various locations pellets of both hormones. It is concluded that testosterone is more efficiently inactivated by the liver than methyl testosterone.

JACOBSEN, Buffalo.

INFLUENCE OF SOME STEROID HORMONES ON LACTATION IN ADRENALECTOMIZED RATS. ROBERT GAUNT, W. J. EVERSOLE and E. C. KENDALL, Endocrinology **31**:84 (July) 1942.

The effects of various steroids of the adrenal cortex and the gonads on the deficient lactation of adrenalectomized rats were studied. There were indications that all substances known to relieve adrenal insufficiency were somewhat helpful, presumably because of their maintenance of a generally improved health. Estrogen and androgen inhibited lactation, even in small doses. Progesterone did not inhibit lactation.

Pellets of estrone were implanted in the spleens of adult ovariectomized female rats, the absorbed estrogen necessarily passing through the liver and there being inactivated. But whenever such animals were placed on a vitamin B complex–free diet protracted estrus occurred. It appears that the liver may have an important part in maintaining the estrogen level of the body within the normal range. Failure to inactivate a portion of the estrogen normally secreted in the body and failure to destroy dietary estrogen as well may be responsible for disorders associated with a high level of body estrogen, such as menorrhagia and metrorrhagia, uterine and mammary hyperplasias and neoplasias, premenstrual tension, some cases of dysmenorrhea, depression of anterior pituitary function and other secondary disturbances. A severe degree of vitamin B deficiency is not necessary for impairment of the hepatic estrogen-inactivating function.

JACOBSEN, Buffalo.

WORK PERFORMANCE OF NORMAL RATS UNDER CONDITIONS OF ANOXIA. SAMUEL S. DORRANCE, GEORGE W. THORN, FRANK H. TYLER and BENJAMIN KATZIN, Endocrinology **31**:209 (Aug.) 1942.

Earlier studies indicated that exposure to anoxia was followed by striking changes in the adrenal cortex. Studies on adrenalectomized rats and dogs suggested that these animals were unable to survive exposures at a reduction in pressure which was well tolerated by normal animals. However, the altitude tolerance of adrenalectomized rats could be greatly increased by appropriate treatment with adrenal cortex extract.

Studies were made on the effect of treatment with adrenal cortex extract, synthetic desoxycorticosterone acetate and amphetamine sulfate on the work performance of a carefully selected group of normal rats exposed to reduced oxygen tension. None of these substances improved significantly the performance of these animals.

JACOBSEN, Buffalo.

RELATION OF THYROID TO GROWTH. MARVIN KOGER, VICTOR HURST and C. W. TURNER, Endocrinology **31**:237 (Aug.) 1942.

Thyroxin-treated mice repeatedly gained an average of 28 per cent more weight during a period of five weeks than did the controls.

Thyroxin-treated mice stored more nitrogen and gained more in body weight per unit of food intake than did controls for the first few weeks of treatment. After the rapid gains of the treated animals had subsided, the controls were more efficient in this respect than treated animals.

JACOBSEN, Buffalo.

PHOSPHORYLATION OF FAT IN ABSENCE OF ADRENAL GLANDS AS MEASURED WITH RADIOACTIVE PHOSPHORUS. N. STILLMAN, C. ENTENMAN, E. ANDERSON and I. L. CHAIKOFF, Endocrinology **31**:481 (Nov.) 1942.

Evidence is offered which establishes the ability of adrenalectomized rats to synthesize new phospholipid molecules.

JACOBSEN, Buffalo.

COMMERCIALLY AVAILABLE NEWER ENDOCRINE PRODUCTS. WILLIAM A. SCHONFELD, New York State J. Med. **42**:1538 (Aug. 15) 1942.

All endocrine products commercially available are classified under various categories. The source, potency and therapeutic uses are briefly discussed.

AIKMAN, Rochester, N. Y.

SYNDROME OF TOTAL ENDOCRINE HYPOFUNCTION. RODOLFO DASSEN, Rev. méd. latino-am. **27**:28 (July) 1942.

In a case of total endocrine hypofunction the author studied its relation to the syndromes of Simmonds and of Falta. The patient was a woman of 26 years weighing 32 Kg. The improvement obtained by large doses of vitamins and extract of the anterior lobe of the hypophysis (25 per cent increase in weight in six months) suggests the influence of avitaminosis on endocrine insufficiency.

DWYER, Kansas City, Mo.

Diseases of the Muscles, Bones, Joints and Lymph Glands

MULTIPLE PRIMARY HEMANGIOMA OF THE BONES OF THE EXTREMITY. Alfred J. ACKERMANN and MAYNARD S. HART, Am. J. Roentgenol. **48**:53 (July) 1942.

This is the fifteenth reported case of multiple primary hemangioma of the bones of the extremity. The patient, a white boy, aged 15 years, entered the hospital with a history of progressive painless swelling of the left ankle and knee for the preceding four years. There was a sunburst appearance of the trabeculae with small loculations.

The treatment of choice of bone hemangiomas thus far has been irradiation. Roentgen rays exert a definite effect on the endothelium of the vascular channels, causing a fibrosis. It is suggested that after irradiation osteoblastic activity of the cells predominates, so that osteoplasia occurs and osteolysis ceases. In cases in which there is a predominance of osteoblastic activity roentgen therapy is not necessarily required. SQUIRE, Chicago.

MORQUIO'S DISEASE ASSOCIATED WITH MENTAL DEFECT. MALCOLM J. FARRELL, JOHN D. MALONEY and PAUL I. YAKOVLEV, Arch. Neurol. & Psychiat. **48**:456 (Sept.) 1942.

Morquio's disease begins at about 1 year of age and progresses for several months. It is characterized by deformity of the thorax, vertebral column and limbs. There are a short neck, dwarfism, kyphosis, flat feet and limitation of motion at some of the joints. The patients in the cases originally reported were of normal intelligence. The author reports 2 cases of the disease with cerebral agenesis. BEVERLY, Chicago.

THE USE OF AGE AT FIRST APPEARANCE OF THREE OSSIFICATION CENTERS IN DETERMINING THE SKELETAL STATUS OF CHILDREN. CLARA C. BUEHL and S. IDELL PYLE, J. Pediat. **21**:335 (Sept.) 1942.

Buehl and Pyle studied the ossification of the distal epiphysis of the ulna, of the sesamoid bone of the thumb and of the crest of the ilium in 30 boys and 30 girls and found that these centers are reliable indicators of maturity. The centers ossify in the order given and mature at 5½, 10 and 13 years in girls and 7½, 12 and 14 years in boys. The age of onset of the menarche in the 30 girls was correlated with the onset of ossification of the three epiphyses. In 66 per cent of the girls ossification of the ilium occurred within six months after the menarche. Buehl and Pyle believe that this ossification in a boy may indicate a point in maturation comparable to that in a girl at the time of the menarche.

Another reliable ossification point is fusion of the distal phalanx of the second finger. This occurred shortly after the menarche in the majority of the group. STETTENBENZ, Buffalo.

SEVERE OSTEOPSATHYROSIS WITHOUT OSTEOPOROSIS. MARTIN REICHLIN, Ann. pædiat. **159**:121 (Aug.-Sept.) 1942.

After an introductory résumé of the literature on the symptomatology of osteogenesis imperfecta, there follows a description of a case of severe osteopsathyrosis. The peculiarity of this case is that roentgen examination revealed primarily no osteoporosis; this appeared secondarily as atrophy due to inactivity. Finally, an attempt is made to explain this feature. It is supposed that with quantitatively slightly reduced metabolism the calcium content may be normal, while the qualitative deterioration of the organic substance causes a correspondingly high mechanical insufficiency. [FROM THE AUTHOR'S SUMMARY]

Diseases of the Eye

TWO CASES OF ORTHOPTIC INTEREST. E. B. ALABASTER, C. RUDD and M. TREE, Brit. J. Ophth. **26**:304 (July) 1942.

The first patient was a boy aged 2 years with left convergent strabismus. Refraction disclosed compound hyperoptic astigmatism. Glasses correcting this error were ordered. Four years later there was a deviation of 12 degrees without and of 6 degrees with the glasses. Orthoptic treatment, including occlusion, was carried out from 1935 until 1937, with only slight change. It was then discontinued. In 1941 the Maddox rod test showed 10 prism diopters of esophoria and the Maddox wing test 11 to 15 prism diopters. There was simultaneous macular perception.

The second patient was a man who had had an injury of the supraorbital margin with a fracture of the skull on the right side. Diplopia was present for two days after removal of the bandages. At the time of ophthalmic examination the right eye was displaced downward 1 cm. and there was slight exophthalmos. A roentgenogram showed a fracture of the right

malar bone, passing obliquely down and out from the orbital margin, and a fissured fracture in both the roof and the floor of the right orbit, passing directly backward from the orbital margins at the sites of an irregularity already noted on palpation. There were 1 degree of esophoria with no hyperphoria when the patient looked ahead, 6 degrees of esophoria with 10 degrees of right hyperphoria when the patient looked downward and fixed with either eye and 2 degrees of esophoria with 1 degree of left hyperphoria when the patient looked up and fixed with either eye. The field of diplopia revealed involvement of the superior and the inferior oblique muscles and of the superior and the inferior rectus of the right eye. Despite the muscular anomaly, the patient, who was engaged in airplane engine maintenance, stated that he ordinarily noticed no diplopia, drove a car to and from his work without trouble, occasionally rode a motorcycle and as part of his work did airplane testing in flight, during which he took over the controls. It must be emphasized that this patient had had no orthoptic training at any time and, in spite of the great displacement of his right eye, experienced only a minor degree of diplopia as a temporary phenomenon. Since then he has been proved to possess stereoscopic vision sufficient for him to have adequate control of a motorcycle, a motor car and an airplane in flight.

W. ZENTMAYER [ARCH. OPHTH.]

Skin Diseases; Allergy

SOME CONSIDERATIONS REGARDING ETIOLOGY OF IMPETIGO CONTAGIOSA. H. S. CAMPBELL, California & West. Med. **57**:136 (Aug.) 1942.

By means of clinical experiments on himself with Berkefeld filtrates of washings or swabbings from active fresh lesions of impetigo with which he was able to produce itching lesions diagnosed as impetigo contagiosa by another dermatologist, Campbell concludes that the causative agent in this disease is a virus rather than staphylococci or streptococci. Lesions produced with primary cultures of staphylococci he believes are really due to a carry-over of the virus, since subcultures fail to effect similar results.

DAVIS, San Francisco.

EXPERIMENTAL REPRODUCTION OF GASTRIC ALLERGY IN HUMAN BEINGS WITH CONTROLLED OBSERVATIONS ON THE MUCOSA. H. M. POLLARD and GEORGE J. STEWART, J. Allergy **13**:467 (July) 1942.

Gastrophic observations were made on four groups of subjects before and after the ingestion of food.

Foods were used to which the patient was sensitive and which produced symptoms referable to the stomach. The first group had abdominal symptoms due to food; the second had asthma due to food but had no abdominal symptoms; the third group had asthma not due to food and the fourth group were considered normal.

The changes produced in the stomach of those having gastrointestinal allergy were distinct hyperemia, lumpy or even nodular appearance, edema and thickening of the rugal folds with diminished peristalsis and grayish mucus clinging to the mucosa. The changes occur mainly in the lower third of the stomach.

HOYER, Cincinnati.

A VEGETABLE MILK SUBSTITUTE: TARO. BENJAMIN F. FEINGOLD, J. Allergy **13**:488 (July) 1942.

Feingold suggests this recipe for the use of children who are sensitive to milk and by whom other substitutes, such as soybean mixtures, are not tolerated.

Ingredients: RECIPE

(1) Poyo meal (taro)	20.0 Gm.	(5½ teaspoonfuls)
(2) Clear gelatin	4.0 Gm.	(1½ teaspoonfuls)
(3) Vegetable oil (olive, cottonseed, corn, sesame)	10.8 Gm.	(3 teaspoonfuls)
(4) Corn syrup	7.5 Gm.	(½ teaspoonful)
(5) Calcium gluconate	2.0 Gm.	(2 teaspoonfuls)
(6) Sodium chloride	2.4 Gm.	(½ teaspoonful)
(7) Water to make	240 cc.	(8 ounces)

Preparation:

Weigh and mix the dry materials. Add the oil, and mix to a paste. Add the corn syrup and water to make 240 cc., or 8 ounces. Cook over the direct flame for three minutes, stirring while cooking. Cool the mixture and restore to the original volume by the addition of water.

HOYER, Cincinnati.

THE VARICELLIFORM ERUPTION OF KAPOSI. A. W. PEPPLE, T. W. MURRELL and R. W. FOWLKES, South. M. J. **35**:667 (July) 1942.

The authors report 3 cases, 2 in adults, in which the lesion may be classified as Kaposi's varicelliform eruption.

In 1 case intracytoplasmic inclusion bodies similar to those of Guarnieri were observed in the epithelium. A keratoconjunctivitis resulted when Paul's test was done.

If the Guarnieri body is accepted as evidence of variola or vaccinia, this lends weight to the belief that Kaposi's varicelliform eruption is a manifestation of vaccinia.

SCHLUTZ, Chicago.

THE TREATMENT OF SCABIES. KENNETH MELLANBY, C. G. JOHNSON and W. C. BARTLEY, Brit. M. J. **2**:1-4 (July 4) 1942.

Because of the large amount of literature which has accumulated on the treatment of scabies much confusion exists. In this article the authors report attempts to use practically all of the sarcopticides that have been recommended and to treat proved scabies with these medicines. They then compare their results and discuss briefly the advantages and disadvantages of the various forms of treatment. The preparations used were 10 per cent sulfur ointment, sulfur vanishing cream, a commercial preparation known as Marcussen's ointment, flowers of sulfur, sodium thiosulfate and hydrochloric acid, sulfur lather tablets and sulfur taken internally. Various derris root preparations were tried as well as benzyl benzoate and a number of less well known preparations.

The experiments reported in this paper indicate that for the treatment of scabies only two substances are satisfactory, and they are sulfur ointment and benzyl benzoate. Of these two agents benzyl benzoate appears to be the better for killing the parasites. However, this is a liquid and cannot be applied to the body satisfactorily by the patients and requires an assistant, while the sulfur ointment can be used by 1 person. BIRDSONG, University, Va.

RELATION OF PYOGENIC SKIN INFECTIONS TO THE SKIN-CARRIER RATE. N. H. MARTIN, Brit. M. J. **2**:245 (Aug. 29) 1942.

This is a brief report of an investigation of the percentage of organisms found at various parts of the body in patients with pyogenic cutaneous infections as compared with those without any cutaneous infections. Two hundred and forty patients were studied. The figures seem to demonstrate that the presence of a localized cutaneous lesion produces a widespread increase in pathogens on the normal skin. The author states: "If this be so it must materially alter our conception of the results to be expected from localized forms of treatment for pyogenic skin infections."

BIRDSONG, University, Va.

Surgery and Orthopedics

SUCCESSFUL CONSTRUCTION OF AN EXTRATHORACIC ESOPHAGUS. JOHN STAIGE DAVIS and EDWARD S. STAFFORD, Bull. Johns Hopkins Hosp. **71**:191 (Oct.) 1942.

This paper is not suitable for abstracting. It is well written and illustrated, and all aspects of the subject are considered. It should be studied by persons interested in this problem.

LYTTLE, New York.

POSTURE HABITS IN INFANCY AFFECTING FOOT AND LEG ALIGNMENTS. H. E. THELANDER and MABEL L. FITZHUGH, J. Pediat. **21**:306 (Sept.) 1942.

Comparison of alignment of the foot and leg was made of 246 children ranging in age from 6 months to 5 years, the majority being between 18 months and 3 years. Neutral position indicated a deviation of not more than 10 degrees either way from the true midposition of hip, knee and foot. Deviation meant a toeing in or out of 10 to 90 degrees. It was found that deviation occurred most often in the knee to chest sleepers, one third of them toeing in and two thirds toeing out (86 subjects). Neutral position was favored by sleeping on the side, and hence it is the most favorable sleeping position. Photographs illustrating various sleeping positions and their effects on position of the foot are shown.

The authors recognize the fact that many abnormal early deviations of the feet spontaneously correct themselves, but they stress that one cannot assume that this will occur. Early recognition of a tendency to deviation, institution of sleeping on the side and use of proper corrective manipulative exercises of the muscles are advised. Pens, cribs, carriages and canvas swings are to be condemned as they tend to limit normal activity and consequently offer a greater chance of foot malalinement. Perpendicular climbing is the best method to balance the muscles.

STETTENBENZ, Buffalo.

THE MODERN TREATMENT OF BURNS. F. McD. RICHARDSON, M. J. Australia **2**:337 (Oct. 10) 1942.

Physiologic changes after severe burns have been ascribed to: (*a*) primary or neurogenic shock; (*b*) loss of water and protein secondary to burns; (*c*) toxic effect of the burn, and (*d*) damage to the adrenal cortex. The primary derangement is loss of plasma from the blood stream, with consequent alteration of the osmotic and fluid balance of the body. The best treatment is replacement of the lost elements. The addition of electrolytes in solution further decreases the effective osmotic pressure in the capillaries, and they will often filter out to increase the already existent edema. The addition of whole blood, while contributing some plasma, inevitably adds quantities of red blood corpuscles to an already overburdened circulation. Two important and simple laboratory guides to replacement and fluid therapy are (1) the hematocrit reading and (2) the protein level of the plasma, which may be calculated from the specific gravity. For patients suffering from severe burns, from wounds, from hemorrhage or from obvious shock these determinations should be repeated every four hours in the early stages and thereafter, as the patient's condition improves, at less frequent intervals.

GONCE, Madison, Wis.

MINOR SURGERY IN CHILDHOOD. H. W. S. WRIGHT, Practitioner **149**:179 (Sept.) 1942.

The author calls attention to the fact that although a minor operation is minor in the eyes of the surgeon, it is a major event in the minds of the child and the parents. He discusses the importance of premedication on the basis of this observation.

Under operations on the upper respiratory tract the author discusses adenoidectomy and tonsillectomy. Inasmuch as these operations are infrequently performed in the office in the United States, this discussion is not as important to the American surgeon as it may be to the English. He advocates general anesthesia and good myringotomy rather than mere "pricking" when the child is suffering from otitis media. For patients with acute lymphadenitis he advocates administration of an anesthetic and a completely effective incision only after pus is present. Other conditions discussed are retropharyngeal abscess, circumcision, meatal ulcer, rectal polypi, anal fissure and prolapse of the rectum.

THELANDER, San Francisco.

Miscellaneous

THE ABSORPTION, EXCRETION AND DISTRIBUTION OF 2-SULFANILAMIDOPYRAZINE (SULFAPYRAZINE) IN MAN. MORTON HAMBURGER JR., J. M. RUEGSEGGER, NORRIS L. BROOKENS and ESTHER EAKIN, Am. J. M. Sc. **204**:186 (Aug.) 1942.

In a study of the absorption, excretion and distribution of sulfapyrazine in the human body the authors showed that the drug is absorbed rather slowly from the gastrointestinal tract. It is excreted more slowly by the kidneys than is sulfadiazine, sulfapyridine or sulfathiazole.

Acetylsulfapyrazine is more soluble in water or urine than sulfapyrazine. Both compounds are many times more soluble in alkaline than in acid mediums.

Sulfapyrazine enters the cerebrospinal fluid slowly, reaching a concentration of about 59 per cent of that in the blood twelve hours after an intravenous injection.

Concentrations of the drug in most body fluids approach or exceed the concentrations in the blood; little sulfapyrazine appears in milk. However, its concentration in the blood plasma is approximately double that in the red blood cells.

HENSKE, Omaha.

SALICYLATE INTOXICATION IN INFANTS AND CHILDREN. HENRY L. BARNETT, JOHN R. POWERS, JOHN H. BENWARD and ALEXIS F. HARTMANN, J. Pediat. **21**:214 (Aug.) 1942.

Five cases of salicylate poisoning are reported. In all instances double or triple the usually recommended dose of salicylate had been given. Clinically, poisoning is manifested by intense hyperventilation, severe acidosis, moderate dehydration, vomiting and stupor, occasionally with convulsions and at times with an acetone odor of the breath. Laboratory tests reveal a low content of carbon dioxide and positive reaction of the urine for ferric chloride persisting on acidification and boiling. Acetone is also present. Treatment consists in the administration of many times the usual amount of alkali given to combat acidosis. Sodium r-lactate (one-sixth molar) injected intravenously gives the best results. In addition, large quantities of carbohydrate should be given, preferably dextrose by intravenous injection since depletion of glycogen is pronounced in salicylate intoxication.

STETTENBENZ, Buffalo, N. Y.

NITROBENZENE POISONING. A. STEVENSON and ROY P. FORBES, J. Pediat. **21**:224 (Aug.) 1942.

This is a report of a case of nitrobenzene poisoning in a 12 month old infant due to a bedbug exterminator spray used in the baby's room. The child recovered. Nitrobenzene is a common ingredient of certain shoe dyes, floor polishes and bedbug sprays and is also found in flavoring extracts for alcoholic liquors, pastries, etc. Cyanosis due to methemoglobinemia is the outstanding feature. Treatment consists in administration of oxygen and intravenous injections of solutions of dextrose. Use of 1 per cent solution of methylthionine chloride is also beneficial. The authors state that the mortality rate is around 20 per cent.

STETTENBENZ, Buffalo, N. Y.

A METHOD FOR THE BIO-ASSAY OF DIGITALIS IN HUMANS. HARRY GOLD, MCKEEN CATTELL, HAROLD L. OTTO, NATHANIEL T. KWIT and MILTON L. KRAMER, J. Pharmacol. & Exper. Therap. **75**:196 (July) 1942.

The method consists in noting electrocardiographic changes in essentially normal subjects. The paper must be consulted for details.

PILCHER, Cleveland.

A TOXICOLOGICAL STUDY OF SUCCINYL SULFATHIAZOLE. A. D. WELCH, PAUL A. MATTIS and ALBERT R. LATVEN, J. Pharmacol. & Exper. Therap. **75**:231 (July) 1942.

Succinylsulfathiazole (2-[N^4-succinyl-sulfanilamido]-thiazole) given in large doses over a period of several weeks has been shown to have a marked bacteriostatic action on the coliform organisms of the intestinal tract of dogs. Preliminary study with effective doses in human beings had shown no harmful actions. The present authors found the drug to be but relatively slightly absorbed from the intestinal tract of laboratory animals, including monkeys. Monkeys were given up to 5 Gm. of sodium succinylsulfathiazole per kilogram by stomach tube every four hours for thirty days without harmful effects clinically or by postmortem examination. Parenteral and intraperitoneal injections also indicated that the drug will not produce toxic actions by oral use.

PILCHER, Cleveland.

EMBOLIA CUTIS BISMUTHICA: AN UNTOWARD ACCIDENT FOLLOWING THE INTRAMUSCULAR ADMINISTRATION OF BISMUTH SALICYLATE IN OIL. F. GOLDSCHLAG, M. J. Australia **2**:144 (Aug. 22) 1942.

Three hours after the injection of a 10 per cent solution of bismuth subsalicylate in oil into the left gluteus muscle the patient began to have severe pain in the buttock radiating into the leg, which was so excruciating on movement as to necessitate rest in bed. Examination two days later revealed an area about 5 by 7 cm. on the left buttock which was infiltrated, swollen, hot and highly tender. The most striking feature was a strange picture formed by blue, slightly elevated, horizontal, parallel bands, about 0.5 cm. wide, distributed on the buttock. A number of vertical smaller and less distinct streaks intersected the main trunks and formed a marmoraceous network, with slight bluish interstices and pronounced, infiltrated, deeply colored meshwork. The acute symptoms disappeared within a few days, but the marmoraceous network persisted up until the time of the report, six weeks later. The pathogenesis of this process is as follows: The bismuth suspension is deposited in a deeper branch of an artery and then carried by the blood stream to the smaller cutaneous vessels, where the needle-like crystals of bismuth subsalicylate form emboli. Clinical symptoms are due to perivascular inflammation and the pain to ischemia.

GONCE, Madison, Wis.

Book Reviews

Synopsis of Diseases of the Skin. By Richard L. Sutton, M.D., and Richard L. Sutton, Jr., M.D. Price, $5.50. Pp. 481, with 413 illustrations. St. Louis: C. V. Mosby Company, 1942.

This book is intended to present in compact form a general view of diseases of the skin. The authors have carefully planned the illustrations, adhering to a ratio of almost a picture a page. The material is presented in a clear, concise manner; thus the volume will be especially valuable for general practitioners and undergraduate students. The book is well organized and, despite its small size, the common and many of the rare dermatologic conditions are described.

Several omissions regarding therapy are outstanding: Sulfapyridine is not mentioned in the treatment of dermatitis herpetiformis, especially in children; bismarsen is not mentioned in the treatment of chronic discoid lupus erythematosus, and sodium iodide is not mentioned in the treatment of herpes zoster. On page 319 there is an erroneous statement that the Plummer-Vincent syndrome is due to a deficiency of vitamin A.

The book is recommended as a valuable addition to the library of the general practitioner and the undergraduate student.

Tuberculosis congenita. By Mario Waissmann. Paper. Price, not given. Pp. 170, with 39 illustrations. Buenos Aires: El Atenéo, 1942.

The first part of this monograph deals with the germinal transmission of the bacilli of tuberculosis and with prenatal contamination through aspiration into a bronchus, or by a hematologic transplantation. This portion is followed by a discussion of the resistance and heredity of the infant organism. The last part of the book describes in detail the 11 instances of congenital tuberculosis in the author's experience. These are fully described and illustrated with photographs of the gross anatomic specimens and photomicrographs. The entire book is well written, and the treatment of the subject seems conclusive. There is an excellent bibliography of Spanish and foreign literature on the subject.

Management of the Sick Infant and Child. By Langley Porter, M.D., and William E. Carter, M.D. Sixth Edition. Price, $11.50. Pp. 997, with 96 illustrations. St. Louis: The C. V. Mosby Company, 1942.

This excellent book has been completely revised in its sixth edition. The subject matter is in agreement with the current thoughts in pediatrics. The writers have reserved all of their illustrations for the chapters on methods of procedures and formulas and recipes. These chapters are not only well augmented by excellent photographs and drawings which amplify the explanation of technical procedures, but they contain an abundance of practical information for the student and the practitioner alike. The book will still rank as one of the leading pediatric texts and fully justifies its sixth revision.

Sindromes de infestación del quiste hidático. By Miguel A. Jaureguy, Instituto de Clinica Pediatrica e Higiene Infantil. Price, not given. Pp. 38. Montevideo: Impreñta Rosgal, 1942.

This is a monograph in Spanish on the syndrome of hydatid infection. The author believes that the cutaneous reaction of Casoni is diagnostic. Outlines of the various symptoms and of treatment are given. An excellent bibliography of seventy-five articles from the South American literature is appended.

Directory of Pediatric Societies

INTERNATIONAL

International Association of Preventive Pediatrics

President: Prof S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

President: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Charles F. McKhann, University Hospital, Ann Arbor, Mich.

Canadian Committee:

Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto.
Secretary: Dr. H. P. Wright, 1509 Sherbrooke St. W., Montreal.
Place: Boston. Time: Postponed indefinitely.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

British Paediatric Association

President: Prof. L. G. Parsons, 58 Calthorpe Rd., Five Ways, Birmingham, England.
Secretary: Dr. Donald Paterson, 27 Devonshire Pl., London, W. 1.

Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.
Secretary: Dr. E. Gjørup, Dronning Louises Børnehospital, Copenhagen.

Nederlandische Vereeniging voor Kindergeneeskunde

President: Dr. J. H. G. Carstens, Servaasbolwerk 14ª, Utrecht.
Secretary: Dr. R. P. van de Kasteele, Laan van Poot 340,'s Gravenhage.
Place: Different places. Time: Three times a year.

Paediatrický Spolok na Slovensku

President: Dr. A. J. C. Chura, Lazaretská 11, Bratislava.
Secretary: Dr. P. Rados, Lazaretská 6, Bratislava.
Place: Pediatric Clinic, University Bratislava. Time: Six times a year.

Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. E. A. Cockayne, 98 Harley St., London, W. 1, England.
Secretary: Dr. R. Lightwood, 86 Brook St., London, W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:15 p. m.

Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

President: Prof. S. Rosenbaum, 26 Bialkstr., Tel Aviv.
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Sociedad Cubana de Pediatria

President: Dr. Angel A. Aballí Arellano, 17 No. 609 Vedado, Habana.
Secretary: Dr. Julio G. Cabrera Calderin, Hospital Mercedes L y 21 (Vedado), Box 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Mercedes, Habana. Time: Last Wednesday of every month.

Sociedad Mexicana de Pediatria

President: Dr. Fernando López Clares, 12/a. Medellín 191, Mexico City.
Secretary: Dr. Jesus Gómez Pagola, Versalles 64, Mexico City.

Sociedad Venezolana de Puericultura y Pediatria

President: Dr. E. Santos Mendoza, Venezuela.
Secretary: Dr. P. Oropeza, Hospital de Niños, Caracas, Venezuela.

Société de Pédiatrie de Paris

President: Dr. B. Weill-Hallé, 49 Avenue Raymond Poincare, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue Pré aux Clercs, Paris, France.
Place: Hôpital des Enfants Malades, 49 Rue de Sèvres. Time: 4:30 p. m.
Place: Hôpital des Enfants Malades, 49 Rue de Sèvres. Time: 4:30 p. m., third Thursday of every month.

Uruguayan Society of Pediatrics

President: Dr. Nicolas Leone Bloise, Avenida Agraciada 2496, Montevideo.
Secretary: Dr. Héctor Mourigan, Avenida Agraciada 1464, Montevideo.

NATIONAL

American Medical Association, Scientific Assembly, Section on Pediatrics

Chairman: Dr. Hugh L. Dwyer, 315 Alameda Rd., Kansas City, Mo.
Secretary: Dr. Gilbert J. Levy, 188 S. Bellevue Blvd., Memphis, Tenn.
Place: Chicago. Time: June 12-16, 1944.

American Academy of Pediatrics

President: Dr. B. S. Veeder, 3720 Washington Blvd., St. Louis, Mo.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.
Region IV. Meeting cancelled.

American Hospital Association, Children's Hospital Section

Chairman: Dr. Joelle C. Hiebert, 299 Main St., Lewiston, Maine.
Secretary: Dr. W. Franklin Wood, McLean Hospital, Waverly, Mass.

American Pediatric Society

President: Dr. James L. Gamble, 300 Longwood Ave., Boston, Mass.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid Ave., St. Louis.

Canadian Society for the Study of Diseases of Children

President: Dr. R. R. Struthers, 906 Drummond Medical Bldg., Montreal.
Secretary-Treasurer: Dr. Elizabeth Chant Robertson, Hospital for Sick Children, Toronto.

Society for Pediatric Research

President: Dr. Joseph A. Johnston, Henry Ford Hospital, Detroit.
Secretary: Dr. Mitchell I. Rubin, 1740 Bainbridge St., Philadelphia.

SECTIONAL

Intermountain Pediatric Society

President: Dr. Eugene Smith, 385-24th St., Ogden, Utah.
Secretary-Treasurer: Dr. W. C. Cheney, 837 Boston Bldg., Salt Lake City.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

New England Pediatric Society

President: Dr. Warren R. Sisson, 319 Longwood Ave., Boston.
Secretary-Treasurer: Dr. James Marvin Baty, 1101 Beacon St., Brookline, Mass.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

North Pacific Pediatric Society

President: Dr. M. L. Bridgeman, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. C. G. Ashley, 833 S. W. 11th Ave., Portland, Ore.

Rocky Mountain Pediatric Society

President: Dr. G. R. Fisher, 23 E. Pikes Peak Ave., Colorado Springs, Colo.
Secretary: Dr. Joseph H. Lyday, 1850 Gilpin St., Denver.

Southern Medical Association, Section of Pediatrics

Chairman: Dr. William Weston Jr., 1428 Lady St., Columbia, S. C.
Secretary: Dr. Angus McBryde, 604 W. Chapel Hill St., Durham, N. C.
Place: Cincinnati, Ohio. Time: Nov. 16-18, 1943.

STATE

Alabama Pediatric Society

President: Dr. Amos Gipson, 948 Forrest Ave., Gadsen.
Secretary-Treasurer: Dr. Ruth Berrey, 2021–6th Ave. N., Birmingham.

Arizona Pediatric Society

President: Dr. Vivian Tappan, San Clemente, Tucson.
Secretary: Dr. Hilda Kroeger, Arizona State Health Dept. (Maternal and Child Welfare Division), Phoenix.

Arkansas State Pediatric Association

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

California State Medical Society, Section on Pediatrics

Chairman: Dr. William C. Deamer, University of California Hospital, San Francisco.
Secretary: Dr. Charles W. Leach, 2000 Van Ness Ave., San Francisco.

Florida State Pediatric Society

President: Dr. Ludo Von Meysenbug, Box 3356, Daytona Beach.
Secretary: Dr. Robert Blessing, 409 Blount Bldg., Ft. Lauderdale.
Place: Concurrent with state association meeting at time of convention.

Georgia Pediatric Society

President: Dr. T. F. Davenport, 104 Ponce de Leon Ave. N. E., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Edward T. Wakeman, 129 Whitney Ave., New Haven.
Secretary: Dr. Herman Yannet, Southbury Training School, Southbury.
Time: Three meetings a year.

Illinois State Medical Society, Section on Pediatrics

Chairman: Dr. Craig D. Butler, 715 Lake St., Oak Park.
Secretary: Dr. A. J. Fletcher, 139 N. Vermilion, Danville.

Indiana State Pediatric Society

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Mathew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

Iowa Pediatric Society

President: Dr. Mark L. Floyd, Children's Hospital, Iowa City.
Secretary-Treasurer: Dr. James Dunn, Davenport Bank Bldg., Davenport.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. A. Clement Silverman, 608 E. Genesee St., Syracuse.
Secretary: Dr. Albert G. Davis, 307 Gas and Electric Bldg., Utica.

Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. Elwood W. Stitzel, 403 Central Trust Bldg., Altoona, Pa.
Secretary: Dr. P. F. Lucchesi, Philadelphia Hospital, Philadelphia.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Charles F. McKhann, University Hospital, Ann Arbor.
Secretary: Dr. Mark F. Osterlin, Central Michigan Children's Clinic, Traverse City.

Mississippi State Pediatric Society

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

Nebraska Pediatric Society

President: Dr. E. W. Hancock, 820 Sharp Bldg., Lincoln.
Secretary-Treasurer: Dr. John M. Thomas, 1102 Medical Arts Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

New Hampshire Pediatric Society

President: Dr. MacLean J. Gill, 14 N. State St., Concord.
Secretary-Treasurer: Dr. Ursula G. Sanders, 46 Pleasant St., Concord.
Time: Twice yearly.

North Carolina Pediatric Society

President: Dr. Thomas M. Watson, Greenville.
Secretary: Dr. Frederick B. Haar, Greenville.

Oklahoma State Pediatric Society

President: Dr. Ben H. Nicholson, 301 N. W. 12th St., Oklahoma City.
Secretary: Dr. Luvern Hays, 108 W. 6th St., Tulsa.
Place: Oklahoma Club. Time: 6:30 p. m., fourth Friday of each alternate month from September to May, inclusive.

South Carolina Pediatric Society

President: Dr. Lonita Boggs, 301 E. Coffee St., Greenville.
Secretary-Treasurer: Dr. Hilla Sheriff, Wade Hampton Office Bldg., Columbia.

Texas Pediatric Society

President: Dr. F. H. Lancaster, 4409 Fannin St., Houston.
Secretary-Treasurer: Dr. John E. Ashby, 3610 Fairmount Ave., Dallas.

Virginia Pediatric Society

President: Dr. Edwin A. Harper, 301 Rivermont Ave., Lynchburg.
Secretary: Dr. Emily Gardner, 1100 W. Franklin St., Richmond.

West Virginia State Medical Society, Section on Pediatrics

President: Dr. Andrew Amick, 1021 Quarrier St., Charleston.
Secretary: Dr. A. A. Shawkey, Professional Bldg., Charleston.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. J. D. Nourse, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. I. B. Silber, 10465 Carnegie Ave., Cleveland.
Place: Cleveland Medical Library Bldg. Time: October, December, February and April.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. I. Nelles Silverthorne, 170 St. George St., Toronto, Canada.
Secretary: Dr. G. P. Hamblin, 2333 Bloor St. W., Toronto, Canada.

Bronx Pediatric Society

President: Dr. Forris E. Chick, 2325 University Ave., New York.
Secretary: Dr. Jacob H. Turner, 121 W. 197th St., New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

Brooklyn Academy of Pediatrics

President: Dr. Harry A. Naumer, 37–8th Ave., Brooklyn.
Secretary: Dr. Lewis A. Koch, 62 Pierrepont St., Brooklyn.
Place: Granada Hotel. Time: Fourth Wednesday of October, November, February, March and April.

Buffalo Pediatric Society

President: Dr. A. Wilmot Jacobsen, 187 Bryant St., Buffalo, N. Y.
Secretary: Dr. Richard A. Downey, 786 Forest Ave., Buffalo, N. Y.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p. m., first Monday of each month from September to June.

Central New York Pediatric Club

President: Dr. Edward J. Wynkoop, 501 James St., Syracuse.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Places: Various cities in New York. Time: Third Tuesday of April and September.

Chicago Pediatric Society

President: Dr. Heyworth N. Sanford, 952 N. Michigan Ave., Chicago.
Secretary: Dr. Henry G. Poncher, 1819 W. Polk St., Chicago.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

Cincinnati Pediatric Society

President: Dr. Lloyd K. Felter, 3144 Jefferson Ave., Cincinnati.
Secretary: Dr. T. Selkirk, 3530 Reading Rd., Cincinnati.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Dallas Pediatric Society

President: Dr. John E. Ashby, 3610 Fairmount Ave., Dallas, Texas.
Secretary-Treasurer: Dr. Gladys J. Fashena, 4585 Belfort, Dallas, Texas.
Place: Bradford Baby Hospital. Time: 1 p. m., second and fourth Saturdays of each month.

Detroit Pediatric Society

President: Dr. Hugh Lewis, Detroit, Mich.
Secretary: Dr. John J. Pollack, 622-26 Maccabees Bldg., Detroit, Mich.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month from October to June, inclusive.

Fulton County Medical Society, Pediatrics Section (Atlanta, Ga.)

Chairman: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.
Secretary: Dr. Harry Lange, 478 Peachtree St., N. E., Atlanta.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: Second Thursday of each month from October to April, 8 p. m.

Houston Pediatric Society

President: Dr. Raymond Cohen, 2300 Caroline St., Houston, Texas.
Secretary: Dr. Betty Moody, 526 Richmond Rd., Houston, Texas.
Place: College Inn, Houston. Time: Fourth Monday of each month.

Kansas City (Missouri) Pediatric Society

President: Dr. Edwin H. Schorer, 1103 Grand Ave., Kansas City.
Secretary: Dr. H. E. Petersen, Kirkpatrick Bldg., St. Joseph, Mo.
Place: Kansas City General Hospital. Time: On call.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Oscar Reiss, 2200 W. 3d St., Los Angeles.
Secretary-Treasurer: Dr. Elena Boder, 1830½ Lucille Ave., Los Angeles.
Place: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd. Time: Second Monday of February, April, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Harry Bikoff, 850 St. Marks Ave., Brooklyn.
Secretary: Dr. Henry Rascoff, 895 Eastern Parkway, Brooklyn.
Place: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., fourth Monday of each month, October to April, inclusive.

Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. Meyeron Coe, 217-02-91st Ave., Queens Village, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144-38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. Harry A. Spigel, 2647 Connecticut Ave., Washington, D. C.
Secretary-Treasurer: Dr. Perry W. Gard, 2520 Woodley Rd., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., fourth Thursday of every month.

Memphis Pediatric Society

President: Dr. F. T. Mitchell, 376 S. Bellevue Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Harry Jacobson, 1193 Madison Ave., Memphis, Tenn.
Place: John Gaston Hospital. Time: Quarterly.

Milwaukee Pediatric Society

President: Dr. John H. Reynolds, 1628 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. F. J. Mellencamp, 324 E. Wisconsin Ave., Milwaukee.
Place: Milwaukee Athletic Club. Time: Second Wednesday of each alternate month, beginning with February.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Edith M. Lincoln, 600 Park Ave., New York.
Secretary: Dr. John D. Lyttle, Babies Hospital, New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northern California Affiliates

President: Dr. Crawford Bost, 490 Post St., San Francisco.
Secretary: Dr. William A. Reilly, 384 Post St., San Francisco.
Time: Second Thursday of September, November, January, March and May.

Northwestern Pediatric Society

President: Dr. Arild E. Hansen, University of Minnesota, Minneapolis.
Secretary-Treasurer: Dr. Albert V. Stoesser, 205 W. University Hospital, Minneapolis.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October

Oklahoma City Pediatric Society

President: Dr. William M. Taylor, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. G. R. Felts, 625 N. W. 10th St., Oklahoma City.
Place: Oklahoma Club. Time: Third Thursday of each month.

Philadelphia Pediatric Society

President: Dr. Theodore S. Wilder, Greene and Coulter Sts., Germantown, Philadelphia.
Secretary: Dr. Sherman Little, 1740 Bainbridge St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday in March, May, October and December.

Pittsburgh Pediatric Society

President: Dr. Carl Ruder, 646 Washington Rd., Mount Lebanon, Pittsburgh.
Secretary-Treasurer: Dr. C. J. Stoecklein, 125 De Soto St., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate month from October to June, inclusive.

Richmond Pediatric Society

President: Dr. Stanley Meade, 913 Floyd Ave., Richmond, Va.
Secretary-Treasurer: Dr. Louise Galvin, 214 S. Boulevard, Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: 8 p. m., third Thursday of each month, except June, July and August.

Rochester Pediatric Society

President: Dr. Herbert Soule, 122 Rutgers St., Rochester, N. Y.
Secretary-Treasurer: Dr. Jerome Glaser, 300 S. Goodman St., Rochester, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee. Time: Third Friday of each month from October to May.

St. Louis Pediatric Society

President: Dr. Jerome Diamond, 508 N. Grand Ave., St. Louis.
Secretary-Treasurer: Dr. Mary A. McLoon, 408 Humboldt Bldg., St. Louis.
Place: St. Louis Medical Society Bldg. Time: First Friday of each month from November to June.

Seattle Pediatric Society

President: Dr. Frederick B. Joy, Stimson Bldg., Seattle.
Secretary: Dr. Sherod M. Billington, Medical Dental Bldg., Seattle.
Place: College Club. Time: Third Friday of each month from September to June at 6:30 p. m.

Southwestern Pediatric Society

President: Dr. Jeanette Harrison, 1136 W. 6th St., Los Angeles.
Secretary: Dr. Henry F. Gallagher, 1930 Wilshire Blvd., Los Angeles.
Place: Jonathan Club of Los Angeles. Time: First Wednesday in January, March May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Campbell Harvey, 35 W. Huron St., Pontiac, Mich.
Secretary: Dr. Harry A. Towsley, University of Michigan, Department of Pediatrics and Communicable Diseases, Ann Arbor, Mich.

Westchester County Medical Society, Pediatrics Section (New York)

President: Dr. John B. Ahouse, 27 Ludlow St., Yonkers, N. Y.
Secretary-Treasurer: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.
Place: Grasslands Hospital, Valhalla, N. Y. Time: Third Thursday in October, December, February and April.

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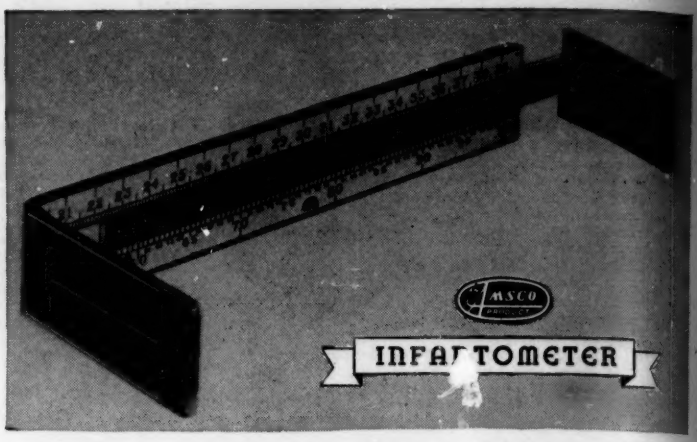

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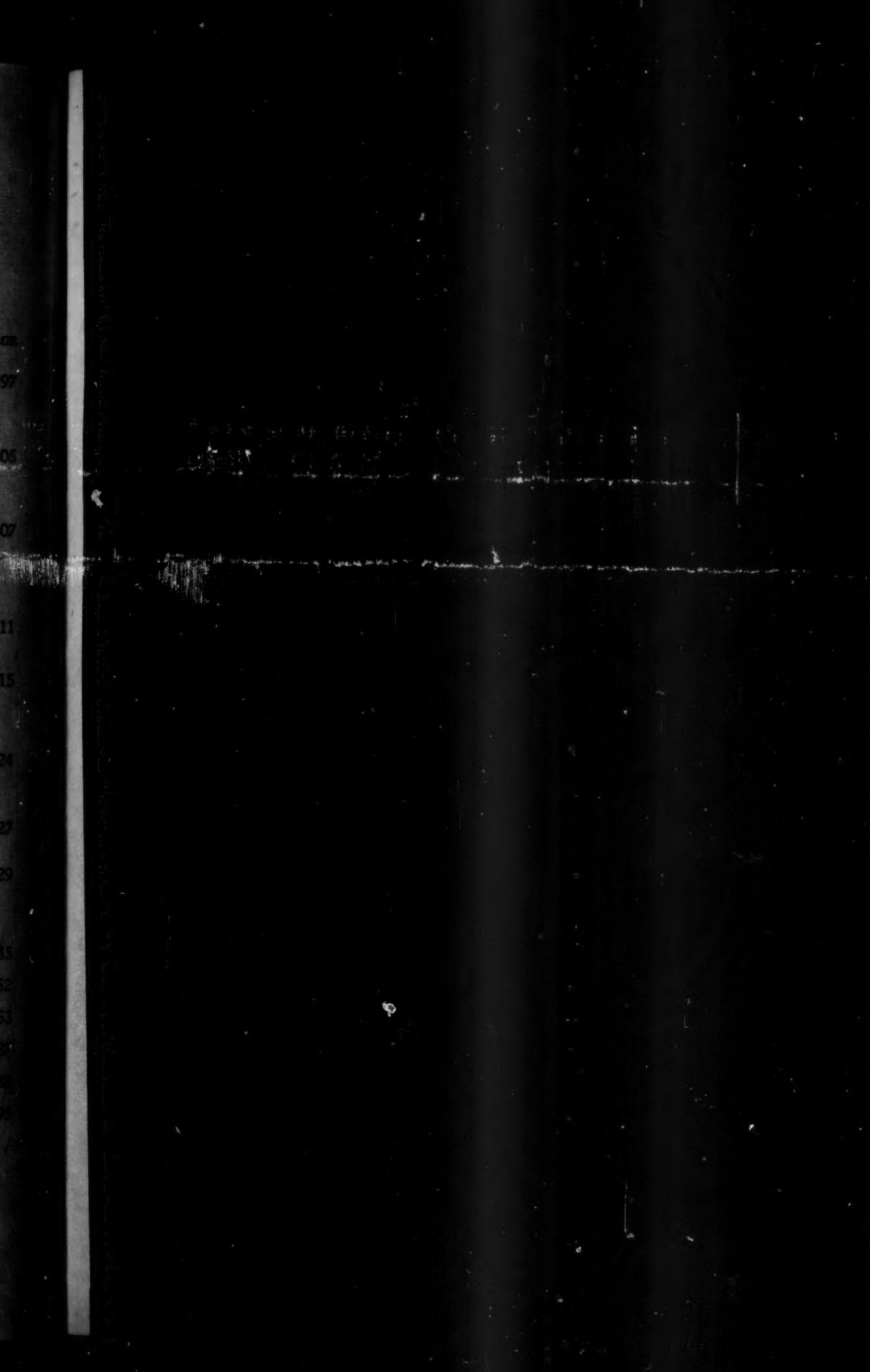
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